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PATHOLOGY

FOR

STUDENTS AND PRACTITIONER'S

An exhaustive bibliography is included in the third volume, (pages 2225 to 2452), with references to the literature of Germany, France, Spain, Italy, Sweden, Switzerland, Denmark, Portugal, Russia, England and the United States.

For greater convenience a complete index is included in each volume and the volume number is printed in italics. The references to illustrations are in bold face type.

While there is in the text a complete system of cross references to related subjects the index summarizes them for ready reference. Example, under the heading "Abscesses" there are 76 entries.

PATHOLOGY

FOR STUDENTS AND PRACTITIONER'S

Authorized Translation of the
Lehrbuch der Pathologischen Anatomie

BY

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IN THREE VOLUMES 1072 ILLUSTRATIONS

VOLUME III

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They are sometimes forced out of the os and hang in the vagina as pear-shaped bodies with the thick end outward, stretched to a great extent. More rarely, cervical polyps are softer and papillary. Their sizes vary; they are seldom larger than walnuts, but may reach the size of hen's eggs or even the size of a child's head. Very often they contain tiny cysts which, in the body of the uterus, are usually small and filled with a thin fluid; in the cervix, where they may reach the size of peas, they are filled with a thick content, in most cases due to retention from obstruction of the excretory ducts. Occasionally there are true cystic polyps. In the cervix, the Nabothian ovulae often have pedicles.

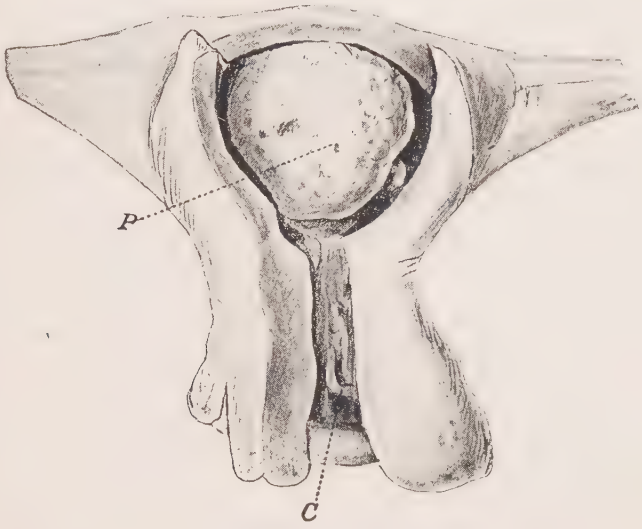


FIG. 824.—Hemispherical mucous polyp P of the posterior wall of uterus with superficial pits and cysts; to left, a smaller one; uterine cavity dilated. Hypertrophy of the funnel-shaped cervix; cervical canal (c) with plicae palmatae; nat. size.

It is not uncommon to see indentations on the surface representing ruptured cysts or wide open mouths of gland lumena. In addition to the cysts of the mucosa of the cervix which are due to inflammatory conditions, there are also cysts of embryonal origin from remnants or inclusions of Gartner's and Muller's ducts; they do not give rise to pedunculated polyps and are surrounded by layers of smooth muscle fibers.

Simple mucous polyps are those in which all parts of the mucous membrane are equally involved. In the so-called adenomatous polyp the glandular overgrowth is predominant; on cross section it appears like a sieve. Other polyps are cystic and others are telangiectatic.

In addition to these benign pure mucous polyps and others, there are also polypoid myomas, sarcomas and carcinomas. The term "polyp" is a gross and not a histological designation.

The surface of a polyp from the body or the cervical mucosa is usually covered with cylindrical epithelium. Those from the vaginal portion of the os are covered by squamous epithelium, and those which arise where the transformation of cylindrical to squamous epithelium takes place are covered by both kinds of epithelium. But polyps in the cavity of the uterus are at times covered by squamous epithelium (Billroth, Kustner, see p. 1625). Metaplasia, as stated on p. 1625, plays an important role in their development.

These changes in the epithelium produce very complicated pictures, easily mistaken for carcinoma. As mentioned before, the cylindrical cell changes to the squamous type by the metaplastic route; it may be observed on the surface, as well as in the glands of the polyps, or the squamous epithelium spreads partly into the lumena of the glands, and partly under its covering of cylindrical epithelial cells, after which the latter disappear, or, finally, it arises autochthonously from indifferent cells. (Studies by R. Oeri, lit.; see also p. 1625, and Hengge and Schauenstein.) There also occur polyps undergoing carcinomatous degeneration on the surface or in the glandular epithelium, or in both (lit. R. Oeri). The first beginning of such carcinomas, because of the complexity of the pictures from the metaplasia and atypical epithelial growth (see page 1625), can hardly be recognized (Benthin). If the diagnosis of carcinoma is made, the picture must be very plain and unmistakable, we might almost say gross (see under diagnosis in uterine carcinoma). Polyps manifest themselves by hemorrhage, often merely an increase of menstruation. But entirely atypical bleeding may appear, finally leading to a high degree of anemia (Gusserow).

The malignant polypoid mixed cervical tumors (see p. 1669) are believed by Wilms to originate from congenital anlage segregated at the time of separation of the germinal layers. Undifferentiated mesoderm and mesenchyme cells are said to be misplaced from their developmental position by the growth of the Wolffian ducts posteriorly into the genitals. Various kinds of tissue arise from these misplaced anlage (lit. R. Meyer).

So-called Erosions of the Os, Erosive Ulcers, and Ectropions.—The so-called erosions of the os are very frequent, mostly end results of cervical catarrh. They are of much practical importance.

An accurate microscopic examination is very important because they occasionally resemble carcinomas, and a suspicious erosion should be extirpated and subjected to microscopic examination.

The usual picture of a so-called erosion of the os is as follows: on one or both lips is a larger or smaller, glistening, intensively red place usually encircling the os and continuous with the mucosa of the cervix; its color contrasts sharply with the gray or bluish red vaginal mucosa of the os;

at times also it lies at a different level. Occasionally they are isolated like islands further away from the os. More rarely, they involve both lips and extend to the tissues of the vagina. Their surfaces are sandy or furrowed, sometimes papillary (fig. 825), the latter easily mistaken for carcinoma.

It might be expected that they are ordinary fresh granulating ulcers, but such is usually not the case.

On the other hand, true ulcers do occur on the os (Fischel) and are called erosive ulcers or granulating or true erosions. The surface epithelium disappears and a uniform granulation tissue covers the surface; the glands disappear; occasionally the uncovered papillary bodies grow forward so that they have a rough appearance. These ulcers are frequent on the surface of prolapsed uteri where trauma is apt to occur during walking, etc., (decubital ulcers); they may be covered by pus or fibrin.

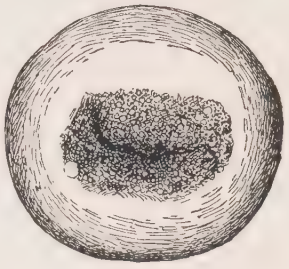


FIG. 825.—So-called erosions of uterine os, chiefly papillary; to the left a small retention cyst at the transition area to pavement epithelium. From a removed uterus, diagnosed carcinoma; nat. size; histologically benign.

In the majority of cases, however, these so-called erosions are of an entirely different character. As J. Veit and Ruge showed, strange to say, microscopically, one layer of cylindrical epithelium is found at the red place, (fig. 826); the stroma of the mucous membrane is very vascular,

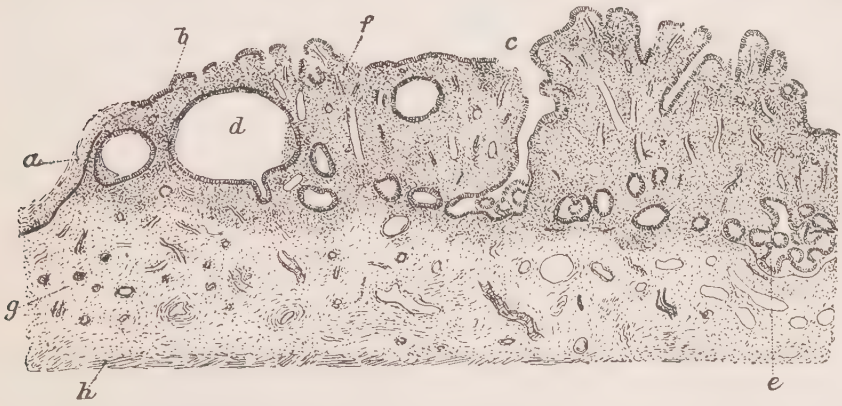


FIG. 826.—So-called erosions of portio vaginalis, partly cystic, partly papillary. Pavement epithelium in several layers at the portio (the deepest layer is cylindrical); b, cylindrical epithelium, single layer; c, gaping opening of a gland; d, retention cyst; e, larger gland, inverted proliferated; f, cellular mucosa tissue with abundant blood vessels; g, vascular connective tissue of the portio; h, smooth muscle fibres. (Med. magn.)

infiltrated by cells and in active growth; there are gland-like depressions of the surface, and important findings are large gland complexes such as are in the cervix. As a whole, these erosions are very similar to an

inflamed cervical mucous membrane, and are markedly vascularized. But there are no ciliated epithelial cells on the erosion. The condition of the glands varies in the individual cases; these influence the microscopic appearance to some extent. There are differentiated:

Simple Erosions.—Smooth or sandy, relatively few small glands in the stroma; papillary erosions: finely granular or honeycombed, many glands distributed as tubes or branching structures closely packed in the depths and these as well as cellular growths of the stroma produce a papillary appearance; glandular: very rich in glands; cystic glandular erosions: porous, spongy with a transparent appearance, many glands dilated and filled with mucus. The cysts are similar in their origin to those in the Nabothian ovula, *i.e.*, retention cysts; scattered examples become as large as peas; the epithelium in the glands is both high and low and piled up in brush and shelf-like projections; many goblet cells are seen.

Follicular hypertrophy is a condition related to adenomas (p. 1672) consisting of cystic glandular hypertrophy of the os. A rounded tumor with uneven tonsil-like surface grows around the os or perhaps only on the anterior lip; in the course of years it may reach the size of a fist, and hang out from between the external genitalia; isolated glands distended with mucus may reach the size of fingers. Inflammatory hypertrophy of the os as a whole may be coincident. An active overgrowth may also occur as in the case of Hofmeier. Peters (lit.) described a polypoid growth of the vaginal portion that was as large as a small pear and appeared similar to a multilocular cyst. The author also has seen structures of this kind as large as walnuts, which microscopically proved to be markedly dilated glands. Henkel believes this cystic hypertrophy of the os originates in some cases from rests of the Wolffian duct, in others as an adenomyositis sero-epithelialis, and in others as Nabothian ovula.

Histogenesis of the So-called Erosion.—The anatomy of the erosion is very clear, but its histogenesis is very much under dispute, especially in relation to the glands. Two main opinions may be mentioned. 1. Inflammatory genesis: (*a*) the cylindrical epithelium of the growing cervical mucosa spreads over the os after its squamous cell covering is macerated by pathological secretions. This process would be the covering of an actual erosion. (*b*) Others, however, assume there are focal erosions of the cylindrical epithelium in the os, the upper layers of squamous epithelium disappearing by maceration from irritating secretion; the lower layers, *i.e.*, the germinative layers, remain and give rise to independent cylindrical epithelium (Ruge-Veit). According to both of these opinions the cylindrical epithelium must be able, after mild stimulation, to grow into the ducts and to produce glandular structures (glands of erosion). But erosion glands may also grow at a time when the squamous epithelium

is still preserved, this from the further growth of cervical glands under the connective tissue of the os; if the surface epithelium is lost (true erosion), the glandular epithelium may cover the denuded surface (gynecologic or so-called erosion). Schottlander (lit.) believes that glands separated in the inflamed cervical mucosa with their excretory ducts obstructed remain in the ducts of the os and then begin to grow actively toward the surface; here they may break through the squamous epithelium, open to the surface, and thus construct the picture of cystic erosion. The author shares this opinion. R. Meyer believes this is a stage of healing whereby the glands are allowed to open through the squamous epithelium; he believes on the whole that the entire so-called erosion process is nothing but a healing stage of true erosion, *i.e.*, ulceration. 2. Embryonal genesis: some authors believe there is persistence of the primary cylindrical epithelium or even of the fully developed cervical structure (one layer of cylindrical epithelium and glands); this persistence is situated on the os which is often excessively large. Others believe these structures are due, not to hindrances, but, on the contrary, to overdevelopment of cervical epithelium during embryonal life (see R. Meyer). Fischel found cervical character to the os in almost 36% of newborn children and called it congenital histological ectropion, (but it seems better to call it so-called erosion). From the sixth embryonal month onward, the cervical mucous membrane crowds the squamous epithelium from out of the cervical canal, sometimes as far as the external os, and on and over the surface of the cervix. R. Meyer calls the process "so-called congenital histological ectropion," or "congenital pseudo-erosion." The rest of the basal squamous epithelial cells under the mucosa increase in number, crawl under the mucous membrane, and later bring it to the top for expulsion. As Orth points out, it is easier to assume that the embryonal glands remain for a time in the depths, even when the outer epithelium has taken on squamous cell characters; if the squamous epithelium later undergoes maceration, the parts underneath immediately show the structure of cervical mucous membrane. By this conception, it is not necessary to consider all of the glands found at the os as pathological new growths (Friedlander). 3. Others believe most of the so-called erosions are really eversions (ectropion) of the lower parts of the cervical canal. There is much about these conditions which remains to be investigated; see Schottlander, Gottschalk, R. Meyer.

The healing of so-called erosions: under the influence of astringents (acetic acid, etc.) there is rapid replacement of the cylindrical epithelium by squamous cells; possibly the former are pushed off by the latter or possibly the squamous epithelium develops from indifferent cells among the cylindrical cells. The glands disappear (Kustner, Hofmeier) because

after the cylindrical epithelium is lifted off they are completely blocked by the squamous layer; then these epithelial depressions are smoothed off with the rest of the surface, and this is the final step in healing (see R. Meyer). After disappearance of the causative cervical catarrh the erosions heal and remain healed, but they do not heal spontaneously with any great readiness.

In both ectropion or eversion, the reddened, softened, swollen, cervical mucous membrane is expelled, even from the virginal orifice, in which case the folds of the arbor vitae are made manifest (inflammatory ectropion or mucous membrane ectropion), or, more commonly, tears ensue, the cervix dilates (particularly in multipara) and particles of the mucous membrane are gradually curled up and expelled to the outside from one or both lips of the os (wall laceration or scar ectropion).

The exposed parts of the mucous membrane are subject to injuries and infections from within the vagina, and are prone to inflammatory secretions and to overgrowths. A rough example is given by the ectropion in prolapse of the uterus. The os uteri is pulled apart and downward by the circular tugs from the vaginal tissues; it descends more and more into the vagina until even the internal os appears at the external orifice. In contrast to erosions, the everted cervical mucous membrane takes on rather an epidermoid (vaginal) character. (See Kermauner, p. 1612, concerning true erosive ulcers in prolapse.) Eversion also occurs in retroflexion and in many carcinomas of the os (Kustner).

Pseudomembranous Inflammations of the Endometrium. –With the exception of during the puerperium, croupous and diphtheritic inflammation rarely occurs and then in severe infectious diseases such as typhoid fever, cholera, scarlet fever, etc.

Infectious Granulation Tumors.– Tuberculosis of the uterus rarely occurs as a primary affair; as a secondary condition it may occur intracanalicularly, by way of the lymph stream from the tubes, through the blood stream coincident with tuberculosis of both tubes and ovaries, from the lungs, or bronchial lymph nodes, or even in general miliary tuberculosis; it occurs in every age, but is rarer in children, (see fig. 819 from a five year old child; see also Graefe, lit.). It is unusual in the pregnant or in the puerperal uterus (lit. by E. Kraus, Schroder). The form taken may be that of flat gray or caseous yellow miliary nodules, often in rows and nests, which become confluent and ulcerate.

Localization of the tubercles is sometimes in the glands, the epithelium of which undergoes overgrowth (v. Franque), but usually they are irregularly scattered in the interstitial tissue. v. Franque has called attention to extensive metaplasia of epithelial cells and irregular layers in the glands and the surface epithelium, particularly in the early stages of

uterine tuberculosis; these findings have been verified by the author, by Alterthum, etc. According to Schottlander, these same pictures in the glands may occur grossly after pregnancy and in gonorrhea. The diagnostic sign is the tubercle, and that alone. The experimental aspect of ascending tuberculosis from the vagina may be found discussed by Jung, Engelhorn, Bauereisen. The ascent may be either through the lymph channels or through the canal (but see the negative findings of Hartmann; see also p. 1598). Rossle believes ascent occurred in a case with extensive

FIG. 827.—Caseous ulcerative tuberculosis of endometrium of the body of uterus (U), miliary nodules in cervix. Caseous tuberculosis of tubes (RT and LT); V, vagina; from a young girl with general tuberculosis; ⅓ nat. size.

ulcerative tuberculosis of the vulva, ulceration of the vagina and caseous pyometra with no involvement of the tubes. See also the literature concerning urogenital tuberculosis.

In other cases, the mucous membrane, especially its upper layers, or perhaps its entire thickness, is ulcerated and changed into a caseous mass of granulomatous tissue (see fig. 827); the cavity of the uterus is filled with a caseous pulp. When the internal and external os are closed the cavity of the uterus becomes dilated to, perhaps, the size of a billiard ball, while the walls are very thin (tuberculous pyometra); this is usually

found in older individuals. On the other hand, both openings may be patulous—the more frequent condition; it is especially in these cases that both the os and vagina are coincidently involved.

In caseous endometritis the purely histological demonstration of its tuberculous character is occasionally impossible, but the finding of tubercle bacilli, often in the serosa, clinches the diagnosis. On the other hand, there is no other change in the endometrium which gives the same appearance, except perhaps a gummatous endometritis which is very rare.

The tuberculosis may spread from the endometrium into the myometrium, but usually in a small percentage of cases. In a case of the author (24 year old woman), a generalized miliary tuberculosis began shortly after childbirth from a tuberculous endometritis. (See also Weil.) In this case the tubercles invaded the walls of blood vessels in the depths of the uterine muscle. Isolated hematogenous tuberculous abscesses of the wall are a great rarity (Amann). The chronic fibrous form of tuberculous endometrium is very rare.

If the cervix is involved, which is rather uncommon, it usually takes the form of miliary tubercles. On very rare occasions the cervix is primarily involved; the author has described a case in a 79 year old woman in whom the fibrocaseous internal surface was similar macroscopically to a slowly growing cavernous carcinoma, but the external and internal os were both intact. (Amann believes this is the only case of primary uterine tuberculosis in the literature.) The cases of Gottsmann and Hofmeier were not autopsied and, therefore, are not conclusive. M. Muller believes primary genital tuberculosis has never been seen. In cases with wide spread tuberculosis elsewhere, the process on the cervix (rare) may resemble a papillary or cauliflower-like growth (Eug. Fraenkel, Glockner, Maly, Chaton, lit,. Everling, Cova, lit.), while the body remains free of the trouble. In a case of Emanuel the prominent tuberculous tumor at the os was mistaken for a sarcoma.

Tuberculous pyometrium as a rule occurs in very old women; the author has seen it in an eighty-one year old woman in whom the atresia was probably a primary senile manifestation.

In a few cases, both tuberculosis and carcinoma have been observed in the same uterus (v. Franque, lit.). The author made this diagnosis from scrapings (fifty year old woman, adeno-carcinoma); in a second case, illustrated in fig. 851, both tuberculosis and carcinoma had deeply infiltrated the cervical tissue; both of these cases have been described by Wallart (lit.); see also Schutze, lit., E. Schmidt. Uterine polyps have also been invaded by tuberculosis (Zahn, Michaelis); the author has observed several such cases.

For the development of tuberculosis in adenomyomas or in adenomyometritis, see Grunbaum, lit., Schutze.

Syphilitic Changes in the Uterus.—Initial scleroses and hard chancres occur on the os, particularly on the anterior lip; furthermore, maculopapular syphilides and gummas also occur in this region.

The soft chancre of non-syphilitic origin is also common in this region.

These findings are very unusual at autopsy, a statement which is particularly true of gummatous lues (see also Mandl, R. Meyer). According to Neumann, about 15% of all the primary lesions are situated in the os; they may be mistaken for sarcoma (Neisser). According to Fuchs, the primary lesion is relatively often hidden in the cervical canal, (spirochetae must be found); see also Liegner. See the illustrations of clinical cases by Oppenheim.

Actinomycosis of the uterus is very rare. It is usually secondary to intestinal involvement, particularly of the appendix. In a case observed at Gottingen (see E. Huffer, lit.), the condition was found in the extirpated uterus of a forty year old woman who had been operated on fourteen days after her twelfth childbirth. The posterior wall was 4 cm. thick and adherent to the surrounding tissues. Extending to the depth of 2.5 cm. was a finely granular, soft tumor, pale, of brittle consistency, and composed of granulation tissue very richly infiltrated by leucocytes, among which were numerous actinomyces. In the case of Verocay, the uterus, from the fundus to the external os, had softened into a spongy meshwork.

Inflammation and Hypertrophic Changes in the Myometrium. Acute Metritis.—In addition to the cases occurring post partum or after abortion, an inflammation between the musculature of the uterus, *i.e.*, in the connective tissue of the interstitium, may occur from infection following injuries such as separations, cuts, crushes, the retention of decomposed secretions after the introduction of tampons, etc., and finally, quite frequently, from gonorrhea (see p. 1624). The uterus swells according to the degree of infection and its localization or its diffusion; bacterial invasion calls forth a cellulo-serous infiltration into the interstitial tissue particularly around the vessels. Interstitial and dissecting abscesses, partial suppuration and necrosis of the musculature, even peritonitis may follow. The myometrium is never involved alone; there is always a coincident endometritis and perimetritis.

Chronic Metritis and Hypertrophic Changes in the Uterus.—By chronic metritis is understood a diffuse hyperplasia of the uterus which, according to the older views, was particularly due to connective tissue overgrowth, but which, according to the newer ideas, is also an hyperplasia of the smooth musculature. It is accompanied by tenderness to

pressure. The enlargement (formerly called uterine infarct) involves the entire uterus or perhaps only the neck.

In the earlier stages, the enlarged uterus is soft, succulent and red in color. The later stages are usually encountered at autopsy; the myometrium is then pale, tough as fibrous tissue and grates on section. The enlargement at this stage is usually minor. The soft stage is due to infiltration into the interstitial tissue of leucocytes and granulation tissue, the hard stage, to the change of these tissues to hard fibrous masses. The end stage of the process is characterized by retraction of the connective tissue and advanced fibrous atrophy of the entire organ. This picture, exactly like that of any interstitial inflammation, is practically always accompanied by an endometritis, very often with erosions, and also by thickening of the peritoneal covering or actual adhesions of the uterus and adnexa (adhesive perimetritis). There is no doubt that in some cases the connective tissue is increased at the cost of the musculature. Labhard believes this is the anatomical reason for post partum hemorrhages. If the change involves the lower uterine segment, rupture may take place during childbirth (Bauereisen).

The viewpoints in chronic metritis are very much in dispute. Most gynecologists do not consider the condition a real entity, inasmuch as they believe the changes in the walls (musculature and connective tissue) are due to individually different evolutions and involutions of the uterus under the influence of age, menstruation and childbirth (Schickele-Keller). Lauth believes "metritic" uteri have hypertrophy of the fibrous and muscular parts proportionate to the relative embryonal conditions, and due to disturbed functions of the ovaries. On the contrary, other authors, *e.g.*, Hofmeier, believe that chronic metritis is an inflammation because of its clinical symptoms (hemorrhages, swelling, pain and tenderness). See the literature by Theilhaber, Shaw, Ahreiner, Hueter, Hirsch (lit.), Pankow, Unterberger, Lauth.

As stated, the diffuse connective tissue hypertrophy and hyperplasia are often accompanied by an hypertrophy of the musculature.

The latter may occur because the uterus does not involute properly after childbirth or abortion. We, ourselves, believe that this is the most important single cause of metritis. Improper involution may occur because of puerperal inflammations in the uterus or in the peri- or parametrium, and furthermore, because of non-inflammatory disturbances of the most varied kind in the puerperium, such as puerperal retroversion, chronic constipation and so on. The processes leading up to this condition are either that the involution is incomplete and fatty degeneration and absorption of the muscle fibers does not proceed to the proper stage, or the regenerative process to replace the destroyed fibers is much more

vigorous than normal; in both cases the uterus is enlarged. If incomplete involution is caused by inflammatory changes, there is a metritis from the very beginning. In other cases, the metritis is secondary in the improperly involuted uterus and may manifest itself by swelling and tenderness.

In other cases hypertrophy of the musculature may occur because of inflammatory changes in the endometrium or in the metrium and subsequent vigorous contractions of the uterus.

Further causes of chronic metritis are: endometritis, particularly of gonorrheal origin, recurring or long continued hyperemias, whether active or passive. Among conditions producing active hyperemia may be mentioned, too frequent coitus, imperfect coitus and onanism, menstrual changes, therapeutic procedures, etc. Chronic venous hyperemia is caused by changes in position, particularly retroflexion and prolapse, by tumors in the neighborhood, to which may be added overfilled bladder and overfilled rectum. When stasis is of long duration, the same changes occur as in other organs, namely, cyanotic fibrous induration (see p. 1628 for the coincident changes in the endometrium).

Pure cases of acquired muscle hypertrophy are found most frequently in connection with tumors, particularly fibromyomas chiefly of the submucous variety, while in interstitial myomas there is an increase of connective tissue in the interstitium (Bertelsmann). It also occurs at times in connection with other tumors such as carcinomas, sarcomas, chorion-epitheliomas, etc., as well as in hematometrias; the tissue becomes thickened and appears laminated just as in pregnancy. The abnormal contents of the uterine cavity and the masses within its walls stimulate contractions and these in their turn produce the true hypertrophy.

Hypertrophy of the Cervix.—The cervix may be divided into three parts; for convenience we use the divisions suggested by Schroeder: (1) the vaginal portion lies wholly in the vagina; (2) the middle portion, only the posterior part of which is in the vagina; and (3) the supravaginal portion is entirely above the vagina.

(See K. Hegar on p. 1708 for remarks on the so-called isthmus of the uterus, *i.e.*, the part between the body and the neck.)

There is a form of hypertrophy of the cervix which is congenital (congenital elongation of the neck). It consists of an elongation, at times so remarkable that the body of the uterus lies in its normal position in the pelvis, while the os appears at the hymen (so-called prolapse without sinking); if the enlargement involves only one, usually the anterior lip, it is called a "snout" hypertrophy. The os may be relatively elongated because of incomplete development of the body. Other hypertrophies of the cervix are the results of inflammation, metritis and cervical catarrhs,

as well as of changes in position of the uterus and the vagina which lead to stasis.

Hypertrophy of the vaginal portion is usually characterized by considerable thickening; it is due to metritis and other inflammatory processes. The thickening may be uniform and diffuse, or nodular. Hypertrophy of the supravaginal portion takes place chiefly in prolapse of the anterior and posterior vaginal walls. Hypertrophy of the middle portion is especially seen in prolapse of the anterior vaginal wall (shortening of the anterior vaginal tissue). The urinary bladder is pushed downward by the hypertrophied middle part of the anterior lip and swells out like a diverticulum. The posterior tissues of the vagina remain intact (fig. 822). The hypertrophic cervix may appear in front of the vulva, and look like an ordinary prolapse. As stated on p. 1614 a true prolapse may also be present.

Inflammation of the Perimetrium, *i.e.*, the Pelvic Peritoneum, and of the Parametrium. Perimetritis or Pelvic Peritonitis.— An inflammation of the serous covering of the uterus is called perimetritis, but in practically every case there is coincident involvement of the neighboring parts of the pelvic peritoneum. Therefore, the term pelvic peritonitis is more descriptive.

The etiology of perimetritis is quite varied. Inflammations of the uterus, the tubes, the ovaries, a parametritis, proctitis, cystitis, even inflammation of the body and muscular parts of the pelvis (coxitis, psoitis in spondylarthritis) may spread to the peritoneal covering of the pelvis. A pelvic peritonitis may also occur from appendicitis. Inflammations which involve the entire peritoneum also involve the pelvic peritoneum.

The character of the inflammation is suppurative or putrid, especially in puerperal and post operative infections; a fatal peritonitis of general distribution follows, or perhaps the process ends in an encapsulated abscess. In other cases, a fibrinous or fibrino-purulent inflammation leads to adhesions between the parts. Chronic productive inflammations are common and lead to fibrous adhesions. Particularly in gonorrheal salpingitis and oophoritis in the later stages, are the uterus and adnexa glued together by avascular veil-like or cobweb-like adhesions.

Misplaced organs where they press upon the peritoneum may give rise to a productive inflammation. Minor changes in the peritoneum causing exfoliation of its covering cells may bring about adhesions between opposing surfaces. If the organ is then brought artificially or spontaneously into its older situation the adhesions become bands; this is seen in retroflexion of the uterus, or in changes of position of enlarged heavy ovaries. Blood coming from a ruptured tube may be encapsulated in the peritoneum (see retrouterine hematocele, p. 1608). New growths may irritate

the peritoneum; local loss of the covering cells, hyperemia, all lead to more or less firm adhesions. If serous exudate is poured out between adhesions, pseudo cystic pictures may result.

Parametritis.—By parametritis is understood an inflammation of the pelvic tissue under the peritoneal covering, not only in the region of the actual "parametrium"—which is found close to the border of the uterus between the leaves of the broad ligament and above the pelvic diaphragm and consisting of connective tissue, fat, muscles, vessels and nerves—but also in the connective tissue between the pelvic diaphragm and the vagina.

Inflammations in this region are particularly common following injuries involving the cervix during childbirth. If this is torn, or if a deep ulcer has appeared, the parametrium may be infected. Or, deep tears may allow entrance through the vagina or the vulva. The spread of the infection, chiefly streptococcic, occurs in the form of a thrombo-phlebitis, a lymphangitis or a phlegmon. The edema and succulence of the puerperal parts open convenient pathways for the spread of infection.

For more details, see under puerperal infections of the uterus, p. 1754.

Non-puerperal parametritis is usually localized strictly around the uterus. It may follow unclean therapeutic procedures on the cervix and in the vagina. Rounded "tumors" appear as large as a child's head. In rare cases, a parametritis follows ulceration of the rectum, appendicitis, or caries of the pelvic bones.

The exudate may be sero-cellular, sero-hemorrhagic, or phlegmonous. The latter may take the form of a soft, edematous swelling with a hard infiltration, rich in fibrin, into the connective tissue, which becomes yellowish or honey-like in color, or it may lead to suppuration and abscess formation with perhaps perforation into the abdominal cavity or into a neighboring organ. When an exudative inflammation becomes chronic it takes on an indurating character and ends in cicatricial thickenings.

(Other histological details by Busse.)

Course.—When the exudative parametritis is fresh and the exudate composed mostly of fluid, complete resorption may take place; cases of longer duration, however, lead to hardenings which pull apart and misplace the pelvic organs. Encapsulated abscesses may renew their activities, even after many years, and the pyogenic organisms suddenly multiply and take on more virulence; perforation may then occur into neighboring organs such as the sigmoid, or a general pyemia may result. In a thirty-two year old woman who had a right multiloculated abscess, the author saw fistulous tracts leading to the sacro iliac synchondrosis, to the right thigh, the right labium majora and into the right hip joint. The chief danger of a parametritis is, however, its spread to the peritoneum.

Uterine Tumors.—The tumor-like polypoid proliferations of the mucosa were mentioned on p. 1632. Lipomas are extremely rare (Merkel, Seydel, lit.), and found chiefly in the body of the uterus: misplacement of fat-forming germinal tissue is supposedly responsible (R. Meyer, Kauffmann). Walkoff described a liposarcoma. In reference to the rare hemangiomas, see R. Meyer, Boks.

Myomas of the Uterus.—Differentiation must be made between (1) the frequent, globular, ordinary myomas or fibromyomas, also called leiomyomas or myoma laevicellulare (formerly "fibroids"), and (2) the rarer adeno types, which, according to Cullen, make up 5% of all uterine myomas; these, in contradistinction to the sharply demarkated myomas, are infiltrating tumors, appearing especially in certain localities (see p. 1657). According to Polano, no sharp clinical separation of these forms is possible, but others call attention to the broad firm adhesions which are common with adenomyomas (comp. Sitzenfrey). (Rhabdomyomas, myoma striocellulare, see under mixed tumors, p. 1670.) Multiple myoma formation in uterine veins, see Sitzenfrey (l.c.) p. 170.

Ordinary fibromyomas are the most frequent of uterine tumors, and occur from the time of puberty onward. They consist of smooth muscle similar to that of the uterine wall, with the addition of fibrous connective tissue. They enlarge from within themselves. They contrast with the uterine musculature by their globular form; the older the growth the more sharply it is outlined; some gyrations, like brain structure, show on their surface (myoma gyratum, R. Meyer). If there is much smooth muscle, the consistency is soft, juicy, and the color has a reddish cast. If the fibrous tissue predominates, possibly because of a minimum development of the vascular supply, the tumor is firm, grates on section, the section surface is striated, white and shiny, the more sensitive muscle fibres cannot keep pace in growth, and probably some fibers undergo atrophy; microscopically, nuclei are less numerous. The connective tissue often becomes partly hyalinized (other degenerations, see p. 1653). In this instance, however, it is not a simple cicatrization process (attempts were made to consider it analogous to scirrhous atrophy of the breast!), for only the large fibromyomas show the marked development of the fibrous tissue, while the smaller ones are more muscular. Cicatricial changes, *i.e.*, connective tissue replacement of disappearing muscle fibers, is but partial, and often only in central parts of the tumors (p. 1653).

The muscle bundles are twisted and matted and consequently on section are seen coursing in all directions. The fibers may thus simulate round cells if the nucleus is just touched, but careful observation reveals their polygonal shape (fig. 828 II). Other cross sections appear without nuclei, since these structures do not all lie in the same plane in any one

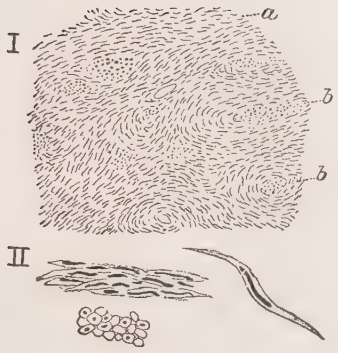


FIG. 828.

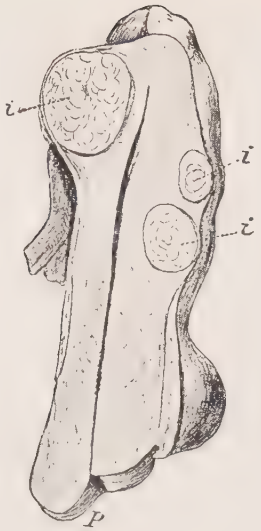


FIG. 829.

FIG. 828.—From a small, soft, gray red myoma of uterus, carmine stain. a, longitudinal; b, cross section; in other places oblique sections of bundles of smooth muscle fibres. (Low power.) II. Longitudinal and cross sections of muscle fibres and one fibre isolated by potass. hydrox. sol. (high power).

FIG. 829.—Small, sharply demarkated firm fibromyomas in wall of uterus cut open by median section; P, os uteri; nat. size.

FIG. 830.—Fibromyoma of uterus. Many cross sections of muscle fibres surrounded by connective tissue (gray). Stain—Van Gieson. (high power).

bundle. The musculature is penetrated by fibrous strands poorly vascularized. Masses of connective tissue separate the various nodules which compose the fibromyoma, and which project on the surface of some tumors as bosses, and everywhere the finest of connective tissue fibrils are found enveloping each single muscle fiber.

Sections stained by the method of van Gieson give a true conception. Pictures are seen as in fig. 830, in which gray masses of fibers and fibrillae, corresponding to the surrounding connective tissue, envelop the light (yellowish brown) muscle fibers, while the nuclei are dark brown.

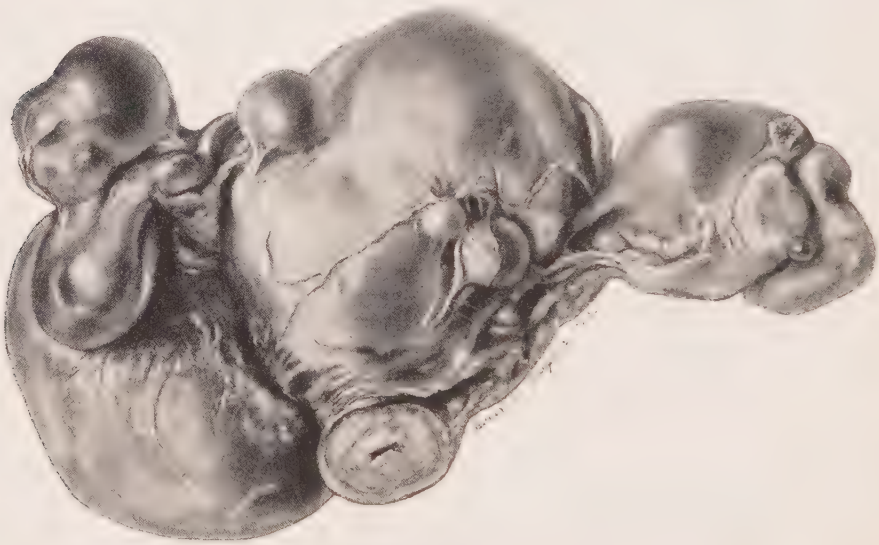


FIG. 831.—Multiple uterine fibro-leio-myomata with both tubes and ovaries. Anterior aspect tilted upwards. Removed at the internal os. This is the ordinary fibro-leio-myoma encountered so frequently in surgical practice. L. H. #18791. ½ nat. size.

In young myomas, still slightly circumscribed, Roesger, Gottschalk and others thought they could demonstrate their development from the musculature of the smallest arteries; according to Cordes, they develop from small swellings of the muscular strands. On the other hand, R. Meyer assumes there is a gradual transition of myomatous to normal tissue, with secondary isolation as to space, perhaps from germ centers not utilized normally (Cohnheim), but not, as Ribbert believes, from atypical cell groups included in the uterine wall. Sakurai claims he found such minute myoma cell centers. Sealone believes he found vascular buds surrounded by indifferent myogenous cells. Faber assumes there is a change in the cells of the uterine musculature. Becher believes myomas begin in swellings of the interlocking muscle fibers (comp. Heimann). The fact that myomas practically never occur in children, but only during the period of adult sexual life, in our opinion, makes the importance of the

"germinal" origin doubtful. H. Freund (lit.) considers it proved that globular myomas may develop from the stimulus engendered by epithelial germinal displacements (see Orloff), after which these germinal factors perish from the pressure caused by the growth of the tumor, and that uteri, deficiently developed and malformed, are especially subject to such myomas (see Herxheimer in reference to myomas as sequellae of developmental interceptions).

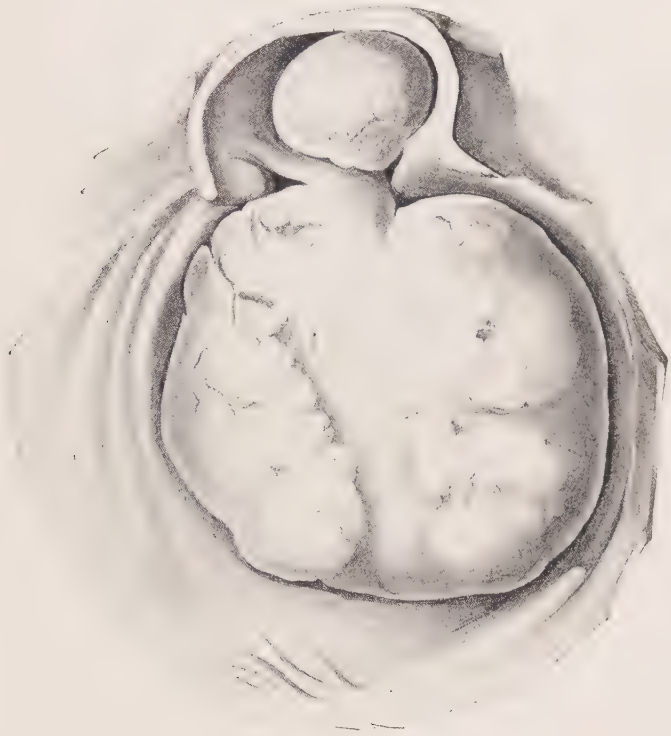


FIG. 832.—Pedunculated fibromyoma of posterior wall of uterus with lumpy, very vascular surface growing downward into the dilated vagina, through pull of the shortened cervix and the almost eliminated os. In the uterine cavity to the left a flat rounded cystic adenomatous mucous polyp; woman 55 years old; ⅔ nat. size.

Microscopically, the differentiation between myomas, spindle cell sarcomas and fibrosarcomas with more markedly developed fibrillar interstitial substance, is not always easy. A mistake for fibroma, the fibrous strands of which are markedly interwoven, is prevented by the use of van Gieson's stain, which shows the extremely delicate reticular structure of the fibrous elements of fibromyomas. (Connective tissue stains red, the muscle yellowish brown.) Connective tissue fibers are wavy; their nuclei are oval or pointed, or tailed. In myomas, in contradistinction to sarcomas, the individual fibers (according to Gebhard 0.2–0.3 mm. long) are arranged more evenly side by side; their fine

fibrous strands are held close together by delicate connective tissue fibrillae. Muscle bundles, on the other hand, show many more nuclei than connective tissue bundles of similar width; the nuclei are long, broader than those of connective tissue, are stretched like small staffs, have rounded ends, and are more uniform than in sarcomas. The nuclei of spindle cell sarcomas are polymorphous, usually thicker, elliptical. The nuclei of neuromas, which in this case are non-myelin, since the myelin forms are well differentiated by Weigert's stain, are smaller, short, ellipti-

FIG. 833.—Numerous globular myomas of uterus, mostly subserous (others intramural and submucous). P, os; RO, right—LO, left ovary; RT, right—LT, left tube; woman 45 years old; ½ nat. size.

cal, and on cross section the fibers are not rounded, but flattened. This differential diagnosis may come in question under skin.

A special difficulty results from the fact that young, quickly growing cellular myomas of medullary, white or grayish white appearance are composed of both short oval to spindle shaped cells. The deciding factor is the uniformity in shape and size of the cells and their nuclei, in contradistinction to the marked polymorphism of cells and nuclei in spindle celled sarcomas, and those myomas undergoing sarcomatous degeneration. In cellular myomas, abundant hyalin connective tissue is frequently seen. If a myoma enlarges during pregnancy, its cells may become as large as those of the pregnant uterus (lit. by Troell).

Isolation of muscle fibers by 33% solution of liquor potassae (fresh solution) is also useful. Immerse 20 to 30 minutes, then tease apart and

mount in the same solution but not in water, otherwise the contractile fibers dissolve completely. If the liquor potassae is at once overneutralized with acetic acid the nuclei can still be stained by carmine. After immersion of one hour in the solution the bundles fall apart and the fibers are isolated.

In well preserved material, special stains (see Benda, R. Meyer, Mallory, Ogata), including van Gieson's stain (see Daels and comp. p. 1655),

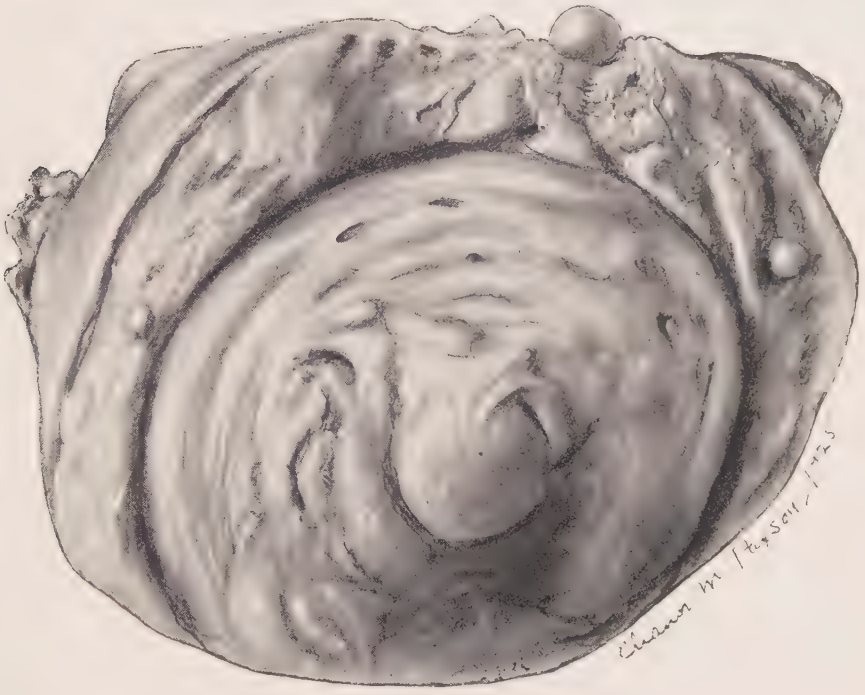


FIG. 834.—Submucous fibro-leio-myoma jutting out of the posterior wall of the uterus and distorting the uterine canal. L. H. #18844. 5/6 nat. size.

reveal numerous fine fibrillae which unite near their poles to larger fibrillae (myoglia fibrillae).

Fibromyomas vary in size from that of a pea to that of a man's head; tumors up to 20 kilos in weight have been observed. They appear singly or in numbers (up to 50). According to their location they are divided into submucous, interstitial or intramural, and subserous; most frequently they are found in the body of the uterus, especially in the posterior wall near the fundus, but may appear near the tubes, and in the vicinity of the internal os; more rarely they are retro- or antecervical (Balaban thinks about 5%), and even more rarely they are at the os (Kolb, lit.).

The soft submucous varieties develop in the layers adjacent to the mucosa and grow into the uterine cavity. At times they bulge hemi-

spherically or become polyps with broad bases or with pedicles (fig. 832). They may cause uterine inversion. Spontaneous expulsion is rare. They are often multiple, and during their growth become more and more distinct from the uterus until they are pedunculated. Torsion may cause total necrosis; torsion of the veins produces hemorrhagic infiltration or "red necrosis;" the pale to dark red color is essentially due to diffusion of hemoglobin (see below). Subserous myomas have been found between the layers of the broad ligament; their pedicles had atrophied, so that they

FIG. 835.—Uterine fibro-leio-myoma with free fluid in the abdominal cavity. Duration four years, but exceedingly rapid growth within two to three months. The clinical diagnosis was ovarian cyst. At operation 96 pounds of fluid and fibro-leio-myoma were removed, the fluid weighing approximately 70 pounds. There was no particular peculiarity about the tumor, nor its position, the accumula ion of fluid not being accounted for on ordinary grounds. The entire peritoneum was thickened, hazy and cloudy, but nothing of a specific nature was detected. Perfect recovery and well being for two years after operation. Further notes are kept as time passes. L. H.

had apparently developed independently. (See Schutze in reference to spontaneous rupture of the capsule of a subserous myoma.) Intraligamentary myomas which grow selectively from the muscular portion of the broad ligament adjacent to the uterus (O. Burkard), and are often deep down in the pelvis, can easily be shelled out, if not too large. Mond reports one weighing 15 kilos. Even moderately sized subperitoneal myomas may cause displacements and flexions of the uterus or compression

of the cervix; larger tumors may distort the body and elongate the cervix. The tube may be stretched over the tumor like a long narrow band. The ovaries are pulled out, elongated, and flattened. Subserous myomas often become adherent to adjacent tissue. Interstitial myomas of greater dimensions often distort the uterine cavity monstrously in length and breadth; at the same time the myometrium undergoes marked thickening and hypertrophy and thus assumes a laminated structure as in pregnancy (see p. 1708). During life this "capsule" may contract spasmodically. Usually a myoma may easily be shelled out from its surroundings (enucleation of myoma). When myomas of different types coalesce, the uterine cavity is extraordinarily distorted. Axis torsion is rare (cervix-torsion of the myomatous uterus, B. B. Schultze, lit., Olow); atresia due to stretching of the cervix, even with subsequent separation of the body from the neck by pure pulling action, has been observed (Hedren, Thorel). See Todyo in reference to dilatation of lymph vessels, to the size of hen's eggs, in the broad ligament. (Omental lymphangiectasias, see Trancu-Rainer.)

Secondary Changes in Fibromyomas.—Inflammatory edema, suppurative infiltration, thrombus formation, softening and necrosis sometimes occur spontaneously during the puerperium, or even, perhaps, after only slight local operative treatment, or in women during the menopause from hematogenous infections (see v. Franque and bacteriological demonstrations by Sitzenfrey). Fatty degeneration and necrosis (gray, clay-colored, or red, with "red necrosis") of muscle fibers are very frequent. The myoma may decrease in size and the firm, fibrous parts remain (fibrous change or induration). X-ray treatments sometimes produce this fibrous metamorphosis. It occurs occasionally in pregnancy (see below), and not rarely during the puerperium when the myoma participates in the puerperal involution of the uterus, chiefly in the form of fatty degeneration (Martin). Dark red myomas undergoing necrosis but showing marked vascularization, thrombus formation and extravasation of blood in the tissues, are called flesh myomas (comp. above). Leith believes that hemolytic bacteria are responsible for the red color due to hemoglobin. Abundant pigment granules are also found. Calcification: the superficial layers calcify or a coral-like scaffolding of lime is deposited; tumors the size of a fist or larger may be completely calcified. The process is frequent in subserous, less frequent in intramural and rare in submucous myomas. The surface is stone hard and shows flat bosses or rolls like brain convolutions. From a woman aged 64 the author shelled out a globular, intramural, completely calcified myoma 17 cm. in diameter, with broad brain-like convolutions. The skeleton of the fibromyoma, illustrated in fig. 837, was as large as a fist in the posterior wall of the uterus of a 78 year old woman. Expulsion of such myomas has been observed (uterine calculi)

even into the bladder or the bowel (lit. by Payr); v. Franque saw beginning expulsion of an intramural myoma into the abdominal cavity (not calcified). Within calcified fibromyomas, sawed apart, are foci of soft

FIG. 836.—Uterus opened anteriorly showing presence of polyps. These were composed of large endometrium-like glands supported in a framework quite similar to the matrix of the endometrium. They were from a patient 40 years old who complained of irregular bleeding for 5 years. The cervical canal is dilated and shows the presence of a chronic catarrhal endocervicitis such as is quite common. The tubes and ovaries showed minor lesions. L. H. #18868. ½ nat. size.

atheromatous or red detritus. After decalcification, (HCl), smooth muscle fibers can sometimes be demonstrated in the calcified areas. Usu-

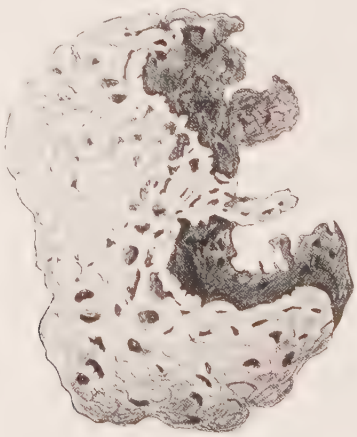


FIG. 837.—Skeleton of an intramural fibromyoma uteri of woman 78 years old; ½ nat. size.

ally, but not always, (comp. Grunbaum, lit.), calcification occurs only in older women. It also takes place after long continued ergotin therapy. True (metaplastic) ossification is rarer (comp. Freund). Lipomas are

rare. For rupture of the capsule of submucous, subserous and intramural myomas, see Schiffmann and p. 1653.

Myomas may enlarge during pregnancy, but according to Opitz, they more frequently diminish in size; perhaps the latter event is due to poor blood supply, to pulling and displacements of vessels, factors emphasized by v. Franque, also for the occurrence of necrosis during pregnancy.

Among the so-called cystic myomas, there are: (*a*) myxomyomas, the connective tissue changes into mucoid tissue and the cells are liquefied. Cavernous spaces without true walls appear, the adjacent muscle bundles

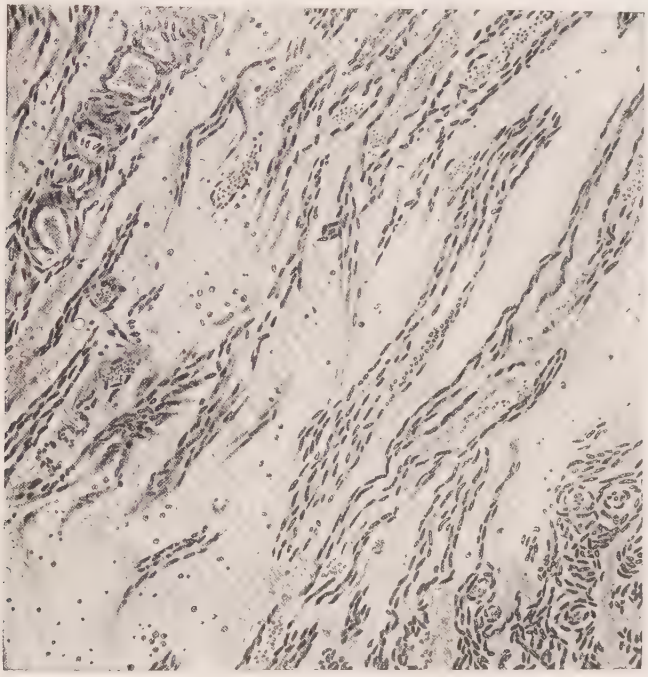


FIG. 838.—Edematous fibromyoma of uterus, from a large subserous tumor showing pseudofluctuation; woman 43 years old. (High power.)

are loosened, torn and fringed, presenting focal firm nodules with prolongations into dense reticulated tissue (comp. Berard and Paviot). Immense fluctuating, transparent and cystic formations may resemble pseudomucinous cystomas. Edematous myomas are similar in appearance; the tissue is saturated with fluid and the muscle fibers undergo atrophy. Softening and dissolving of the muscle cells are plainly seen, and their fibrillar composition (myofibrillae) is well demonstrated by van Gieson's stain. Finally a gelatinous, yellow liquefaction takes place. Edema may be caused by torsion of pedicles (lit. by Piquard), as the author saw in a pseudofluctuating, sanguinous, edematous myoma twice the size of a

man's head, and attached to the posterior wall of the cervix by a finger's breadth of tissue. Unilocular edematous myomas containing several liters of fluid are rare (pseudocystoma edematosum, comp. Loewenstein). (*b*) Soft myomas undergo fatty degeneration, necrosis, and dissolution of muscle fibers until soft areas and finally hollow spaces are developed.

(*c*) In lymphangiectatic myomas, the honeycomb spaces are partly lined with endothelium and contain lymph. Beckmann describes one of this kind weighing 30 pounds (lymph tracts of myomas, see Polano, Schleicher, Leith).

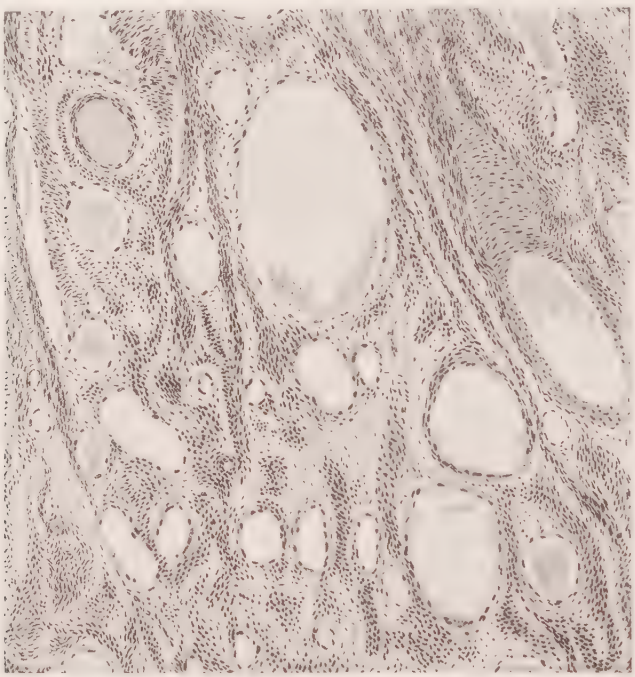


FIG. 839.—So-called teleangiectatic, or cavernous fibromyoma of uterus, from a blue red intramural tumor as large as a fist; woman 50 years old. (High power.)

(*d*) In telangiectatic or cavernous myomas, in contradistinction to the common characteristics of fibromyomas in which there are but few capillaries and many muscular arteries undergoing secondary changes, there are numerous wide capillary blood spaces occupying a part of, or occasionally, the entire tumor. Thrombosis occurs in these vessels. The veins of the uterine ligaments are often enormously dilated (as thick as a thumb). Schlagenhaufer (lit.) described such a case with pure myoma metastases in the liver and lungs (myoma malignum). There have occurred but isolated similar cases.

(*e*) Individual cystic hollow spaces may be caused by hemorrhages and subsequent alteration of the extravasated blood.

(*f*) For adenocystomas or cystadenomas, see the following. Comp. also Frankl in reference to cystic myomas.

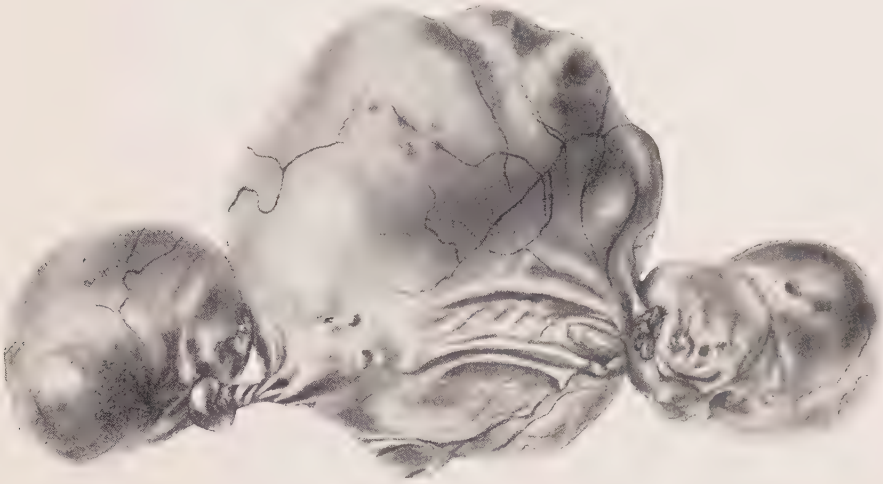


FIG. 840.—Uterine fibro-leio-myoma. Both ovaries cystic. The pedicles twisted. This is an anterior aspect of this unusual condition. Surgical specimen. L. H. #18966. 3/8 nat. size.

The **adenomyomas** include infiltrating tumors of varying size composed of smooth muscle and gland inclusions. These latter are either directly surrounded by muscle fibers or are imbedded in a cellular tissue

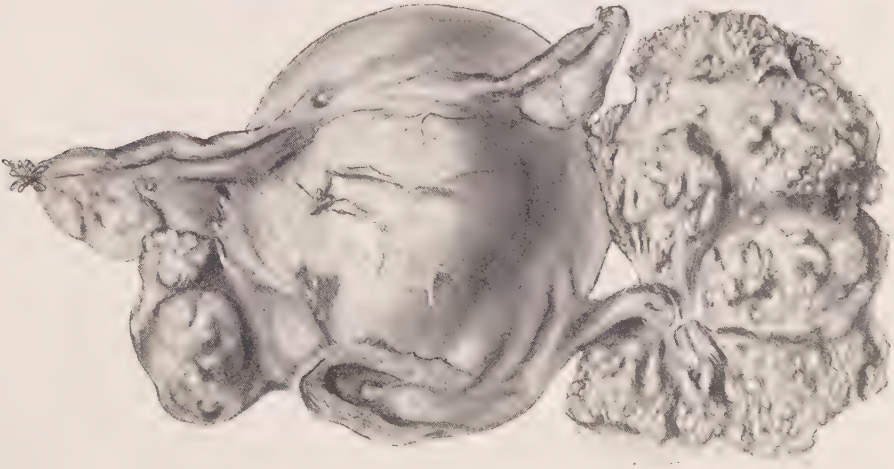


FIG. 841.—Calcified uterine fibro-leio-myoma. Calcification occurs, if not in large amount, at least in small amount in more than 25 % of fibro-leio-myomata. This particular example required a saw for its incision. It is attached by a pedicle in which there is a very meagre blood supply. Possibly this initiated the changes which led to subsequent calcification. L. H. #19062. 1/2 nat. size.

suggesting endometrium, perhaps infantile, to which the muscle bundles are attached on the outside. (Fig. 844.) Cyst adenomas or cystic myo-

mas may develop by dilatation of the glands from secretion. The relation of glandular and myomatous components varies, and sometimes the adenomatous character may predominate, (adenomas). In other cases the epithelial inclusions are merely incidental.

Macroscopically, cross sections of the adenoma, which has usually caused considerable thickening and infiltration of the uterus as a whole, show soft angular circumscribed sunken islands of grayish red color

Fig. 842.—Suppurating myoma of the uterus. This tumor grew in the cervical region. The cervix itself was lacerated from a previous pregnancy and showed chronic ulceration. The tumor was soft and juicy and exuded pus from innumerable pin-point abscesses scattered throughout its substance. The suppurative process was localized to the tumor and no other parts showed extension. The clinical diagnosis was carcinoma because of the purulent discharge admixed occasionally with small amounts of blood. L. H. #567. ⅞ nat. size.

between the silky sheen of the strands of the fibroadenomatous portions; within them are many rounded or elongated cystic clefts. These islands are partly adenomatous and partly cystic, embedded in the ground tissue which is like endometrium.

The source of the epithelial inclusions within the myomas is not always the same. In some, the mesonephric adenomyomas, (according to L. Pick), the origin is said to be (1) rests of the primitive kidney (v. Recklinghausen, comp. Schickele, O. Ernst, lit.); but at present this conception

is practically abandoned. It was assumed that certain parts of the Wolffian bodies, *i.e.*, the paroopheral parts, had become incorporated into Müller's ducts (pp. 1464 and 1568). As positive microscopic proof, there is the combed arrangement of gland ducts corresponding to those of the embryonal primitive kidney and its remains, parovarium and epididymis, as in the ampulla-like main channel containing the openings of numerous, approximately parallel, lateral branches with cyst-like ends. The

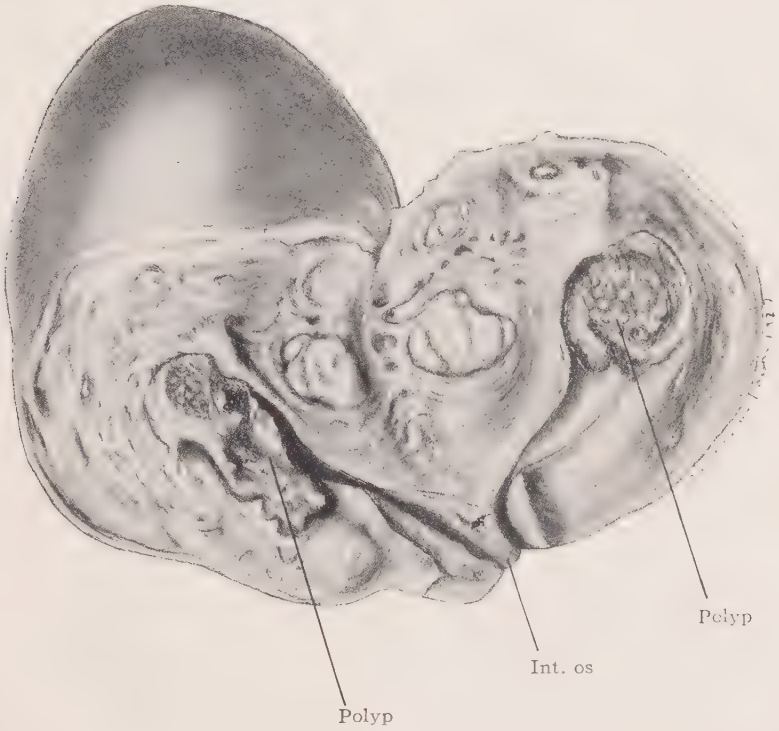


FIG. 843.—Fibro-leio-myomatous uterus opened to show the presence of a large glandular polyp situated in the fundus. The entire uterine musculature in this case was "myomatous" but there were distinct nodules embedded in it as shown in the illustration. Amputation was done at the level of the internal os. L. H. #946. ⅔ nat. size.

epithelium is partly ciliated. It was also pointed out that pigment granules appear in the lumena of the canaliculi and within the stroma, and that so-called pseudo-glomeruli are found as vascular, globular or hemispherical elevations in the end clubs and tube ampullae; they were considered equivalent to involution forms of glomeruli of the primitive kidney, described by A. Czerny as the organ of Giralde (see p. 1461). In reference to the relations of the muscular to the epithelial constituents, *i.e.*, whether both are derived from the ducts of the epioophoron, which also has a muscular sheath, or whether the myoma develops in response to the stimulus of the glandular elements, the question is answered by v. Recklinghausen in

the latter sense. L. Pick and R. Meyer assign a secondary role to the muscular part and do not consider it an integral factor in the beginning and development of the tumor, while Kossmann and Ricker refuse to accept this theory. R. Meyer came to the conclusion voiced before by v. Franque, that the morphology of all adenomyomas of v. Recklinghausen's "paroophoral type" thus far met, is not conclusive in proving or disproving the mesonephric origin of the epithelium; although of course

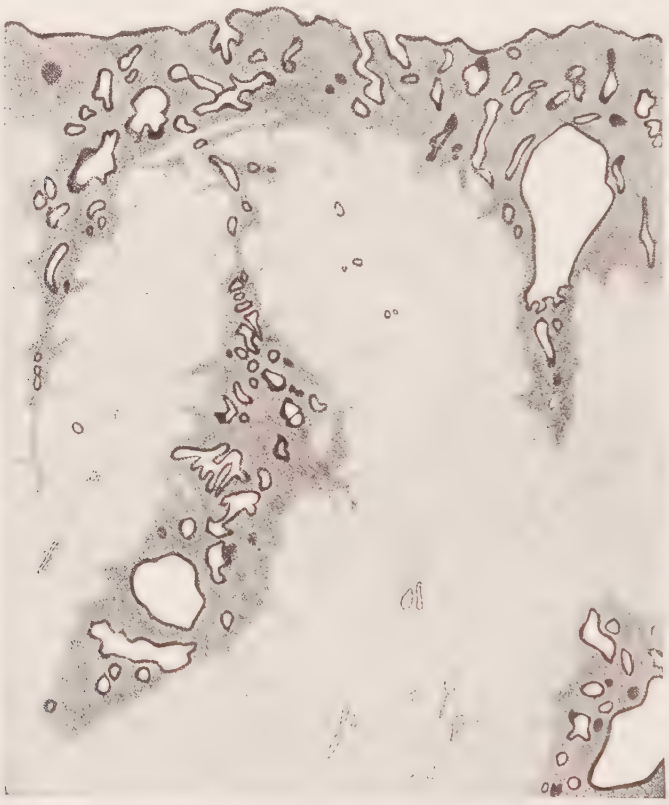


FIG. 844.—Adenomyometritis (so-called mucous adenomyoma). Above, mucosa with surface epithelium and glands; epithelial projections with glands, some dilated deep in the myometrium; thick walled uterus as large as a good egg; removed on account of hemorrhages; woman 65 years old. (Low power.)

the possibility is by no means excluded of tumor development from paroophoron germ cells in the uterine ends of the tubes or in the uterine cornu or in the fundus, (v. Recklinghausen's sense). (R. Meyer in E. IX 1905, discusses this matter more in detail as against the dissertations of Schickele; see O. Ernst also.) Cullen refused to accept the idea of mesonephric adenomyomas (comp. Kelly and Cullen's monograph *re* uterine myomas) and R. Meyer stated in a later paper that the hypothesis

of primitive kidney genesis of adenomyomas of the uterus and tubes is applicable probably only in the rarest of cases.

According to v. Recklinghausen, the positions of the primitive kidney adenomyomas are selectively in the dorsal uterine wall, especially in the peripheral layers (extension to the rectum, see p. 1664), or quite scattered, even penetrating the deepest layers as central tumors. At the tubular angles they likewise are said to prefer the dorsal side and the outer layer. He explains these preferences by the situation and rotation of Müller's ducts at the crossing place of the primitive kidney when fragments become incorporated into Muller's duct, or in the tubes themselves lateral from their angles. Schickele believes he saw misplaced elements of the primitive kidney system in cyst adenomyomas.

Inguinal adenomas are also said to be derived from the germinal elements of the primitive kidney (adenofibromyomas and sometimes only adenofibromas). As mentioned on p. 1464, they are connected with the ligament of the primitive kidney, *i.e.*, the round ligament. In reference to these and other almost exclusively benign tumors of the round ligament, see lit. by Emanuel and Chevassu who found in them a renal glomerulus (see also Frankl). The critical review by Weishaupt (lit.) accepts isolated cases as evidence, but as to the rest, favors the view of origin from serosa epithelium from inflammatory causes. The occasional adenomas of the true ovarian ligament are also included here (lit. by Sitzenfrey), as are those of the broad ligament (see Avoni, resembles paroophoral structure), and those situated below and on the posterior vaginal vault (comp. Schickele); but these latter belong rather to the Wolffian duct (see below).

(2) For other cystadenomas and adenomyomas, rests of the Wolffian duct (Gartner's) may be accepted as starting points (Breus). They are most frequent in the cervical region, where the duct has outward bulgings and gland and duct-like blind branches. These findings are mostly accidental, *e.g.*, the case of O. Burchhardt; exceptionally, they are large enough to demand operation *e.g.*, the case of G. Klein. Most of them develop toward the peritoneum and into the broad ligaments. Adenomyomas of the vagina near the vaginal vault may also be derived from the Wolffian duct (v. Herff. Pfannenstiel, Pick).

In view of all these opinions concerning the paroophoral adenomyomas of the uterus (see under (1) p. 1658), it is remarkable that they are not more frequently observed in the localities which contain rests of the primitive kidney, *i.e.*, in the broad ligaments starting from the epoophoron (see pp. 1464 and 1569). This fact was explained according to Waldmeyer's views, by the dissimilar differentiation of epoophoron (remains of the sexual) and paroophoron (remains of the elementary secretory primitive kidney factors).

Justification for this physiological separation is however doubtful (Aschoff). Pick afterwards found an autochthonous adenomyoma of the broad ligament with all the primitive kidney characteristics (see p. 1659) which every paroophoral adenomyoma shows, but this was derived from the epoophoron.

Perhaps the primitive kidney elements enclosed within the ovaries possess the faculty of producing adenomyomas (lit. by v. Babo).

(3) Without doubt, Müller's ducts, *i.e.*, their gland-containing mucosa, must be considered very seriously as giving origin to these growths; thus, by abnormal inward proliferation of the glands or fetal sprouting of the epithelium of Müller's duct in the angle of the tube, or by multiple germination of Müller's tube epithelium (v. Recklinghausen, Ricker, R. Meyer); by embryonal displacement of germinal mucosa (R. Meyer, Pick), *i.e.*, of glands and cellular tissue; by embryonal scattering of germinal gland tissue (Babes, Diesterweg, Hauser); by post embryonal pinching off of uterine glands (C. Schroeder, Rugi), or by new formations on the part of glands of the mucosa (R. Meyer) which develop by constant growth or extension from the endometrium into the musculature. This latter group has been named mucosa adenomas or mucosa adenomyomas. They occur in the uterus, and, as Cullen correctly emphasizes, they form the bulk of adenomyomas; but they are likewise encountered in the tubes, and according to Tucini, in the broad ligaments also. It is a disputed question whether "adenomyomas" of the tube, for which most authors claim an inflammatory origin (comp. p. 1594), originate much more frequently in this way than from mesonephric displacements (comp. v. Franque; see also Santi). There is no law in relation to the form and localization of these uterine adenomyomas (comp. L. Landau), but the central layers of the corpus uteri seem to be favored. Amos described a decidual metamorphosis in the mucosa foci of such an adenoma.

In typical mucosa adenomyomas, a tumor-like enlargement of the uterus occurs and the organ becomes much thickened. (The author has seen uteri weighing 900–1000 gms.) There are also observed partly congenital and partly acquired gland formations (R. Meyer), surrounded by cellular connective tissue and extending from the mucosa deep into the uterine wall; these justify the appellation of gland-containing mucosa plugs (R. Meyer, Pick, Stein, Pforte, Ruge, I). It is a diffuse disease of the uterus, often accompanied by only moderate enlargement and caused by inflammatory influences; it is called adenomyometritis or adenomyositis uteri. The author investigated a typical case in Breslau in January 1898, in a 50 year old woman who also demonstrated the clinical symptoms of the disease (comp. Opitz); *i.e.*, since her 13th year she had constant hemorrhages and after the menopause (46 years) she had irregular, sometimes

dangerous, metrorrhagias. After repeated curettements over a period of 4 years it was decided to extirpate the uterus. The inner surface of the enlarged firm uterus showed macroscopic crypt-like indentations, which corresponded to areas in which the gland-containing mucosa has sunk deep into the myometrium almost to the serosa; the glands were isolated, small elongated tubes, more rarely several with branchings. There was also deep cystic dilatation; the cavities contained blood and pigment. The epithelium was composed of a single layer. Soon afterwards, the author saw a uterus with uniformly thick walls removed from a 52 year old woman with hemorrhages and suspected carcinoma. The corpus showed crypt-like depressions everywhere. Such cases are correctly classed with the mucosa adenomyomas with relative preservation of the normal form of the uterus (Cullen). Others, *e.g.*, Schutze, speak of "diffuse adenomas," although it is not easy to conceive of "a tumor" when only a thick-walled uterus is seen. According to Cullen, the mucosa itself enters the clefts of the primary diffuse myomatous tumor. But to the author, it is much more plausible that there is a primary growth of the mucosa from inflammatory irritation—for isolated glands without stroma are also seen,—and that under guidance of the epithelium there is reactive hyperplasia of the myometrium. This condition is the perfect counterpart of the so-called "nodular salpingitis" (p. 1593). R. Meyer thinks the mucosa-like stroma is formed by hyperplasia of fixed connective tissue cells due to inflammation; it might be spoken of as adenomyometritis (adenomyositis uteri). Frankl, on the other hand, does not believe there is an endometritis, but thinks that there is primary diminution of reaction of the musculature against the ingrowth of glands. The hemorrhages would then be due to the kinking of vessels within the myometrium and not to hormones. The author saw such cases in old women in whom there were also pedunculated mucous polyps in the uterine cavity.

Sitzenfrey described multiple miliary submucous myomas as small nodules in the mucosa from the size of hemp seeds to lentils. They had developed on the base of an adenometritis, and were the cause of uterine hemorrhages that could not be checked. In the uterus from a 40 year old woman with the wall thickened up to 6 cm. and more, Frankl saw thick fringy polypoid proliferations of the mucosa (resembling corpus carcinoma), into which numerous muscle bands had proliferated.

Finally (4), adenomyomas may be derived from serosa epithelium which has proliferated into the myometrium (heterotopic) after metaplastic change into cylindrical epithelium. These tumors reach considerable size (lit. by R. Meyer), especially when on the dorsum of the uterus, for although they share the microscopical appearance of the mucous adenomyomas, they are contrasted by their position in the outer layers

(see p. 1662). The designation "adenomyoma" might with advantage be changed to "adenomyometritis" or "sero-epithelial adenomyositis" even in these cases, (R. Meyer), because the development of the heterotopic epithelial proliferations, as a rule, is due to inflammation, to which adhesions, etc., all call attention. These serous epithelial proliferations are also responsible for uterine subserous cysts (see v. Meyenburg), and are

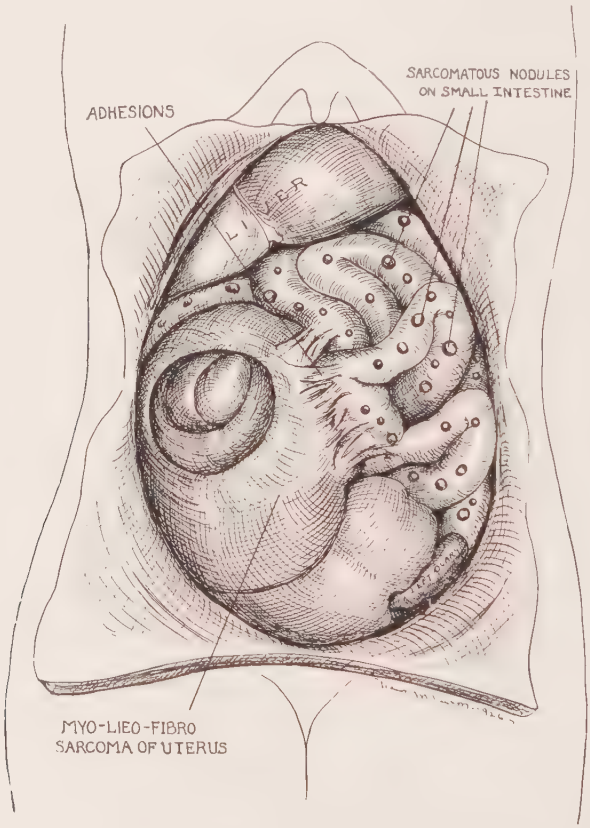
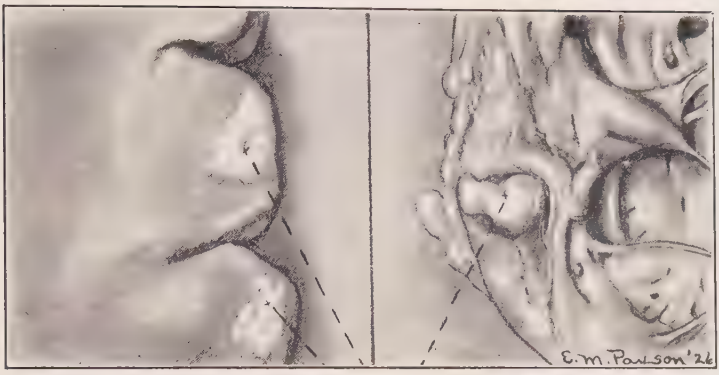


FIG. 845.—Showing the position of a fibro-leio-myo-sarcoma of the uterus with metastases to the intestines, the heart, spleen, the diaphragm, pleurae and lungs. L. H. #130.

well known in the peritoneum (see p. 875) and in serous adenomyomatosis of the bowel (p. 806). Adenomyosis of the rectum and uterus or vagina may suggest a stenosing tumor of the rectum, and has often been treated surgically as such (see, among others, the cases of Renich, Weishaupt, Becker, and R. Freund, lit.). Heterotopic tumor-like serous epithelial overgrowths may spring from other places, such as the tubes (see p. 1590), ligaments, from the parietal serosa of the abdominal wall in abdominal scars (see p. 882) and after ventral fixation, all due to inflammatory processes. See p. 1667, extension of a benign adenomyometritis to the rectum.



Spleen Heart

Under surface of diaphragm

FIG. 845.—(*Continued.*)

Sarcomatous degeneration of a fibromyoma is not rare.

According to Roessle, the designation "deterioration" should be replaced by "degeneration." Histological judgment is not always easy; myomas which grow more quickly, or have very cellular areas, may easily be mistaken for sarcomas. Two factors should be considered: (*a*) with the continued growth of myoma tissue it does not produce "normal" smooth muscle cells, but sarcoma cells, and their spindle shape deviates more and more from the shape of muscle cells; the nuclei become thicker and shorter, ovoid, polymorphous, sometimes there are giant nuclei; they are frequently hyperchromatic and show nuclear divisions (direct and indirect). The cells are closely crowded without interstitial substance, are short and plump; large numbers of giant cells are common. Finally the sarcoma cells lose all similarity to muscle cells. They are differentiated from quickly growing myomas, and also from the common pure spindle cell sarcomas, by the fact that their cells are smaller and poorer in protoplasm. It is also thought that occasional indifferent cells are hidden in the myoma germ cells and that they later become the starting point for sarcomas. Transition to sarcoma may be seen in many places and finally everywhere in the myoma. Since the sarcoma cells are the lawless aftergrowth of muscle cells, the process is not metaplasia, but degeneration. This sarcomatous degeneration of the myomatous components of a fibromyoma or a "pure" myoma is its most frequent form (see among others v. Kahlden, Williams, Morpungo, Pick, Mastny, v. Franque, especially R. Meyer, also Hertel, Evans, etc., see below). The nomenclature varies: with Williams, the author calls them "sarcomatous myoma;" the myoma undergoes sarcomatous degeneration; the name "myosarcoma" is also used (see Raab, Ogorek, lit.). On the other hand, it is wrong to say "myomatoid sarcoma" (R. Meyer), which really means a sarcoma which is, or becomes like, a myoma. (*b*) A sarcoma may arise from the fibrous component of a fibromyoma (Ricker, v. Franque, Hansen, Zacherl, Hoevels), in which case it should be called "myosarcoma," since there are myomatous and sarcomatous components; it might also be called "fibromyosarcoma." These are generally spindle or round cells, or myxosarcomas. Malignant myomas produce metastases by the blood channels, possibly through the lymphatics also; therefore biologically, according to their growth, they are malignant, but histologically (not decisive) they are typical myomas (see p. 1650), distinguished from sarcomatous myomas in that their muscle cells are more uniform and of higher development. For no good reason, Raab doubts the occurrence of such cases (see Lahm).

According to Winter (lit.), malignant degeneration is most frequent in submucous myomas and becomes more frequent with advancing years (v. Kubinyi). Sometimes only isolated areas are found; sometimes the

exact center of an older fibromyoma shows sarcomatous degeneration. The author saw this repeatedly in myomas which macroscopically were not suspicious, and for this reason he considers these changes not unusual. (This has been verified by R. Meyer.) Evans (lit.) found it 72 times, *i.e.*, 1.8% among 4000 operated fibromyomas; Leith calculated about 2%, (see Proper and Simpson, Rive, Dahlet, Rerreitter); numbers like 10% (Warnekioss) are much too high (comp. Raab). That it actually is degeneration of an older, originally pure fibromyoma, can be recognized by the marked change of shape of the uterus, the distortion of the cavity and the hypertrophy of its walls, as in any simple intramural myoma of large dimensions and long existence. Perhaps other fibromyomas, still pure, may be found. Old calcified portions, especially if in the periphery of the tumor, may still be preserved. But luxuriantly growing sarcoma nodules appear on the outside of the invaded uterine wall. Sarcomatous areas are fairly homogeneous, glassy, vascular (occasionally hemorrhagic), light gray, reddish, grayish yellow, or perhaps milky white, and often show pseudo fluctuation. Soft, partly necrotic and hemorrhagic intramural sarcomas may rupture into the uterine cavity; tumor fragments become separated. A thick soft sarcomatous polyp, attached to the main tumor, may hang down into the dilated uterine cavity as far as the cervix.

According to observations of Seitz and Wintz, sarcomas react to the x-rays quite differently from fibromyomas. While the latter regress very slowly, possibly because of cessation of ovarian activity, sarcomas are quickly and directly affected and disappear in a few weeks. Partially sarcomatous fibromyomas shrink more slowly.

Carcinoma in myoma nodules is occasionally an embolic metastasis (Schaper, Schmorl), or an invasion from a carcinomatous peritoneum, or, more frequently, of a uterine carcinoma. v. Winiwarter points out that a sarcomatous degeneration of the stroma of the carcinoma may simulate a carcino-sarcoma. Carcinoma may also develop from the epithelium of an adenomyoma. Thus the rare cases can be understood in which a muscle-invading adenocarcinoma appears in the uterine wall (v. Recklinghausen and others). The author saw the uterus of a 57 year old woman contain both adenomyometritis and adenocarcinoma of the body. Sitzenfrey describes multiple squamous cell carcinoma nodules of the glands in addition to adenomyometritis of the uterus. The author saw an intramural adenocarcinomyoma of the posterior wall of the body (64 year old woman) which had infiltrated the outer layers of the rectum as well as produced a scirrhous, retracting infiltration of the posterior pouch of Douglas (Rolly, later Dillmann, Schwab, Cullen, Polano report similar cases). It is well to remember that a histologically benign adenomyometritis of the posterior uterine wall may extend to the adjacent

rectum (lit. by Sitzenfrey, R. Freund), and that some of the cases of recto-uterine adenomyositis belong in this group (see p. 1667).

An extremely rare counterpart to this, a sarcomatous adenomyoma of the uterus, was seen by the author in a 72 year old woman. The tumor, the size of a man's head, was composed in part of simple fibro-myomatous tissue which had undergone myxosarcomatous change. It also contained scattered fragments of cartilage and bone. Its interior was penetrated by gland ducts resembling uterine glands and in many instances surrounded by soft, endometrium-like tissue (see Iwanoff, Bauereisen, R. Meyer; comp. Froeschmann). Lindquist described tuberculosis in a large diffuse adenomyoma of the posterior uterine wall.

Sarcomas of the Uterus.—Sarcomas of the uterus are relatively rare, and in proportion to carcinomas are about as 1 to 50.

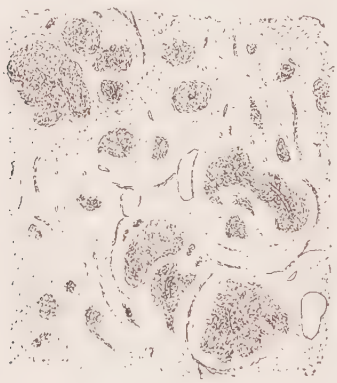


FIG. 846.—Hemangiosarcoma of uterus; currettings from fundus. Spindle-cell tumor masses in dense cloaks around blood vessels almost occluding them (magnifying glass).

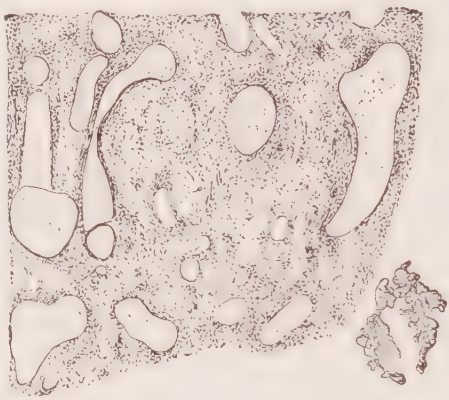


FIG. 847.—Enchondromatous myxosarcoma of uterus; thick gray red, soft tumor fragment, with delicate nodular masses of cartilage visible and palpable in the currettings (magnifying glass). b, Macroscopic cartilaginous masses from another case of chondrosarcoma uteri (see text).

1. **Differentiation.**—The more frequent sarcomas are those of the body springing from both the endometrium and the interstitial tissue as diffuse growths; therefore in the beginning they are differentiated with difficulty from inflammatory hyperplastic proliferations. Hemangiosarcomas also are observed, see fig. 846. Extension is usually diffuse, polypoid, lobulated, coxcomb-like, or grape-like. Tumors the size of a man's head have been seen. In other cases, the quickly disintegrating tumor invades the uterine wall at once and grows through it, whereupon the peritoneum becomes covered with sarcoma nodules. In both cases, detached glassy tumor fragments may be passed per vagina. The myometrium is frequently hypertrophic.

Sarcomas springing from the uterine wall are partly derived from interstitial tissue or from blood vessels, and partly, most frequently in fact,

from muscle cells, not only from the mature forms, but also from immature embryonal muscle cells (myogenous and myoblastic sarcomas). The mature muscle cells may be uterine wall cells or the myomatous components of a fibromyoma. Myomas with sarcomatous degeneration are at first similar to other myomas (see p. 1666). Primary wall sarcomas are often more firm, nodular, and rarely spread diffusely (fibrosarcomas). Parietal and mucous sarcomas may be combined. Sarcomas may also penetrate myomas.

(Lit. by Gessner, R. Meyer, with sarcomatous degeneration of a fibromyoma; see p. 1666 and lit. in appendix.)

Sarcomas of the body of the uterus may grow through its wall and invade the vicinity. Peritonitis, perforation of bowel, even perforation of the abdominal wall, have occurred. Often, however, the tumor remains localized for some time and in contradistinction to carcinoma, the uterine adnexa are usually undisturbed. Generally speaking, metastases are not very frequent; when seen, they are especially in the lungs, liver, vagina, skeleton (see fig. 643, p. 1225). From a sarcoma of the body of the uterus which itself was immense, we saw metastases as large as a child's head to both ovaries.

Secondary tumors have not infrequently been observed in the vagina, and in the immediate vicinity of the uterus, *e.g.*, at the urethral meatus. Discontinuous nodules, especially in the vagina, may arrive per blood stream, but are considered by many as implantations. Recurrences after operative removal may show the most extraordinary and diffuse dissemination. It seems sometimes that the prognosis is less favorable in sarcomatous fibromyomas than in endometrial sarcomas.

2. **Sarcomas of the cervix** are much less frequently found (according to Gessner the proportion is 8:2) and are polypoid, lobulated, or grape-like; the latter is rare in the body of the uterus (Keitler, lit. Frankl). They are soft, partly edematous, partly myxomatous, partly lymphangiectatic spindle cell sarcomas, at times, with giant cells (lit. Borrmann). Those of the cervical mucosa show a tendency to hang into the vagina and to become necrotic (mistaken for mucous polyps!). They are extremely malignant on account of their rapid growth and quick recurrence (lit. Peham, Backer and Minich).

Sarcomatous polyps also occur in the os uteri. Ehrlich described a sarcoma of the portio with the macroscopic appearance of a carcinoma. Adenomyxosarcomas of polyp-like nature are rare in the cervix. In reference to cross striated musculature in the os—and in the rare corpus sarcomas, which occur in children, (and also in extreme age), and the theory of their origin, see under vagina; lit. by Kehrer, Berka and by Glynn and Bell. An unusual type of tumor, easily mistaken for carcinoma,

is called "lymph endothelioma," derived from the endothelium of lymph vessels and spaces (Amann, Pepere, lit. by Kirchgessner, Sell). They occur, among other places, in the cervix, and occasionally in the body of the uterus; they are malignant. Only if the transition of normal endothelium can be positively proved (which may be difficult) can carcinoma be absolutely excluded. The author has never seen such a case (see also Sperber and R. Meyer). It is well to remember that minor proliferations of lymph vessel endothelium occur here and there in cervical sarcomas and also in uterine fibromyomas. Sometimes both sarcoma and carcinoma have been seen in the uterus (lit. in appendix), either side by side, or secondarily combined, or indeed growing from the very beginning as mixed tumors—sarco-carcinomas, or further, from the sarcomatous change in the stroma of a carcinoma (carcinoma sarcomatodes). The author demonstrated many years ago that sarcoma-like areas in carcinomas may be simulated by diffuse extension of very polymorphous carcinoma cells. For liposarcoma, see Sitzenfrey. As Pick first showed in a child 2½ years of age, (see also P. Hoffmann, lit.), the sympathetic embryonal ganglioma (see p. 1288) also occurs in the uterus, and can easily be mistaken for a round cell sarcoma, or a globocellular carcinoma.

Microscopically, uterine sarcomas are usually of the round or small or large spindle cell types; round and spindle cells are often found together, or spindle cells of varying sizes, thickness, and nuclear structure resemble sarcomatous myomas. To these are added isolated giant cells, or even numerous, polymorphous giant cells with gigantic lobulated nuclei (as in the case of Fellander, lit.). A myxomatous change, even cyst formation, develops here and there in the tumor tissue and, more especially in the grape-like forms, a hydropic swelling due to blood and lymph stasis; fatty degeneration is common. In reference to sarcomatous degeneration of myomas, see p. 1666. Sometimes cross striated muscle fibers are found (lit., Lawen, Grieger, Glynn and Bell); or cartilage in sarcomas of the body of the uterus (lit., Hunziker, Pietzold) and more frequently of the cervix (see p. 1634). In hemangiosarcomas, several subtypes can be distinguished, among them perithelial (Gottschalk) and other perivascular sarcomas (fig. 846), or intravascular endotheliomas which are easily mistaken for carcinomas (case of Hansen and Beckhaus), and angiosarcomas with hyalin degeneration of the vessel walls. (See fig. 959 under tumors of the spinal meninges which have also been called cylindromas.) R. Meyer displays much scepticism in reference to angiosarcomas. Polano (lit.) described an angioblastic sarcoma. Telangiectatic sarcomas have been reported. Melanosarcomas are rarities. In sarcomas of the body of the uterus, but more often in the grape-like cervical type, macroscopically visible cartilaginous masses occur between and through the vascular

and cellular sarcomatous tumors (fig. 847); they look like transparent, bluish red, delicate, nodular, coralline areas. (Seydel in his case calls it embryonal connective tissue, from which differentiated cartilage and spindle cells!) On microscopic examination the cartilage is hyalin. These arise from misplaced germinal fragments (according to R. Meyer mesodermal germ cells from which cartilage springs by a metaplastic process) or by metaplasia of sarcoma tissue, *i.e.*, young connective tissue, and are also found in the metastases (they have been mistaken for fetal remains of an abortion!). In the curettings from a case of sarcoma, the author found cartilaginous masses which could be recognized with the naked eye (fig. 847-b); the uterus was removed soon afterward, and showed remnants of a diffusely growing sarcoma of the endometrium with cartilage inclusions, and an intramural sarcomatous degenerated fibromyoma the size of a small apple in the posterior wall. No metastases were found at autopsy of this extremely anemic woman. Fig. 847 is another case. The case mentioned on p. 1668 belongs to those with cartilage inclusions, but such chondrosarcomas are quite rare (see Gebhard, Duchinoff, lit. Puech and Massabuan, Gaebelin, Pietzold, lit.). Penkert describes a teratoid mixed tumor which revealed a carcinoma of the body of the uterus with myxedematous and cartilaginous stroma. Lymphosarcomas are seldom found. Schlagenhaufer reported a case in which the starting point was probably preexisting adenoid tissue of the os, *i.e.*, where true lymph follicles are found in the mucosa. The same author reported cases of lymphogranulomatosis with involvement of the adnexa, and one case of chloroleukemia with a green uterus (good illustrations).

Microscopic diagnosis of sarcoma may be difficult in curetted material if it is not angiosarcoma with its characteristic architecture—especially so, since the normal, and far more the cellular inflamed endometrium, resembles sarcoma. It should be made a rule to obtain knowledge of the clinical data in any case. The tissue must be embedded carefully in celloidin or paraffin, and thin sections cut, in which possibly the topographical relation can be recognized. As far as possible the low power should be used; if high power is used on single cells, as is the usual custom with beginners, it is easy to diagnose a sarcoma in merely inflammatory hyperplastic endometrium, *e.g.*, in a myomatous uterus, or in an endometrium with carcinoma of the os. The points for recognition are the finding of hyalin cartilaginous masses and giant cells. Angiosarcomas cannot be mistaken, as stated above. In a slide of hyperplasia of the endometrium in the premenstrual stage which can be topographically recognized, various levels are seen with abundant cells and glands, but with fairly regular arrangement; thus there is a lower spongy layer and a more compact upper layer with elements resembling decidua cells (see p. 1619).

If there is a mucous hyperplasia (p. 1629) the architecture remains well preserved in spite of the increase of blood and cells in the interstitial tissue (gland interstitial tissue, surface epithelium), just as in true inflammation of the endometrium with round cell infiltration (see p. 1629) or with abundant spindle cells in the interstitial tissue (p. 1628). Sarcoma of the endometrium is quite different. There is complete penetrating proliferation of the mucosa by rounded, spindle shaped or polymorphous cells which are not evenly spread nor of uniform staining power; the glands are absent or present as mere traces, and the surface epithelium has disappeared; or the cells may not be arranged in layers in the same section but in irregular mixtures of dense cellular spaces consisting of round or spindle cells and reticular areas, some well stained and others stained poorly or not at all, *i.e.*, necrotic. If, on the other hand, glands are found embedded everywhere in the ground tissue, even if the interstitial tissue is very cellular and suggestive of sarcoma, as it may be, if high power is used, there is no sarcoma which could have developed from endometrial ground tissue. Mistakes for abortions must not be made; their large pale poorly circumscribed decidual cells with pale nuclei and still more, the chorionic villi, are quite characteristic.

Adenoma of the Uterus.—In discussing diffuse and circumscribed inflammatory hyperplasias of the endometrium in the body of the uterus and cervix it was pointed out that some glandular inflammatory proliferations of the mucosa are on the border line of tumors, *i.e.*, of adenomas. The same holds true for some gland-containing erosions. Follicular hypertrophy of the os (Schroder), mentioned on p. 1636, with its glandular proliferations, is so close to the border line of inflammatory to adenomatous neoplasms, that some class it with adenomas (for instance B. Winckel). Comp. Hartmann and Lecene.

Labhardt described a diffuse adenomatous proliferation in the cervical tissue which he thinks was a congenital hypertrophy of the uterine mucosa.

We reserve the designation "adenoma" for such epithelial tumors which consist only of glands and their scaffolding supporting the vascularization. Polypoid adenomas are frequently seen as benign glandular tumors confined solely to the mucosa (adenoma simplex), usually of the cervix.

Menge described a polypoid, entirely benign, "uterine adenoma" (advanced age), which becomes noticeable by atypical hemorrhage and sanguinous discharge.

Diffuse adenomas may be confined to the mucosa (adenoma simplex) and, as such, occur in advanced age. They are, as Schroder emphasizes, similar in certain ways to the hyperplastic processes (see p. 1629), but they show a tendency to malignant degeneration and thus become adeno-

carcinomas, or the so-called destructive or malignant adenomas of the gynecologist—a malignant tumor, often villous, broad based or diffuse, which crosses the mucous border, and had better be called a glandular carcinoma. It is merely a highly organized form of adenocarcinoma.

Carcinoma of the Uterus.—With Ruge and Veit, we distinguish, according to their starting points, (1) carcinomas of the os, (2) of the cervix, and (3) of the body of the uterus; (1) and (2) together are also called cervical carcinomas.

Carcinomas of the os are the most frequent, and occur particularly in women who have had children. They usually start from the surface epithelium and are made up of squamous cells; they may either infiltrate and cause an immense thickening of the whole cervix or of one lip, or form papillary proliferations and cauliflower-like shapes.

Uterine carcinomas are quite frequent. According to the data at the institute of Basle, and Gottingen, they occur in 14.75% and 15.59% respectively, of all cases of carcinoma. Only occasionally are they observed in children (lit. Engelhorn, Glockner, P. Hoffmann). Bumm-Aschheim report a cervical carcinoma (adenocarcinoma) springing from the posterior lip, and filling the whole vagina, in a suckling, 7 months old.

FIG. 848.—Carcinoma (K) in posterior lip of os uteri (HM) with an ulcer crater (G); anterior lip (VM); entrance into cervical canal (Ce); two small myomas in uterine wall; median longitud. sect.; $\frac{3}{4}$ nat. size.

Carcinomas of the os are histologically of the same character as squamous cell carcinomas of the outer skin. Some show an abundance of keratosed carcinoma plugs (epithelial pearls) in which giant cells also occur (lit. Moench, Obata); others show the characteristics of the so-called basal cell carcinomas (see under skin), and others cylindrical, or spindle-shaped cells due to firm pressure. The large plugs are often extensively necrotic, markedly infiltrated with leucocytes, sometimes resemble plump glands and show no keratinization. The author has repeatedly seen masses of eosinophile cells; in fact, they are usually present in large numbers in the uterus with carcinoma. The same is true of plasma cells (see W. Fischer, Weishaupt). If Schottlander and Kermauner have not seen carcinomas of the basal cell type, it can only be understood that they

mean the classical penetrating gland-like squamous epithelioma (see pictures under skin). The solid type, by far the more frequent, is certainly not rare, as Krompecher emphasizes.

Some are adenocarcinomas, starting either from eroded glands (see Keitler) or from deeper glandular portions (p. 1636); a deep nodule develops in one lip and causes marked distension with later rupture (fig. 848). The lip may become truly cavernous. Papillary carcinomas (cauliflower growth) are usually composed of squamous cells and rarely of cylindrical cells. Superficial suppuration is frequent. There are also benign papillary growths found at the os, such as papillary erosions or

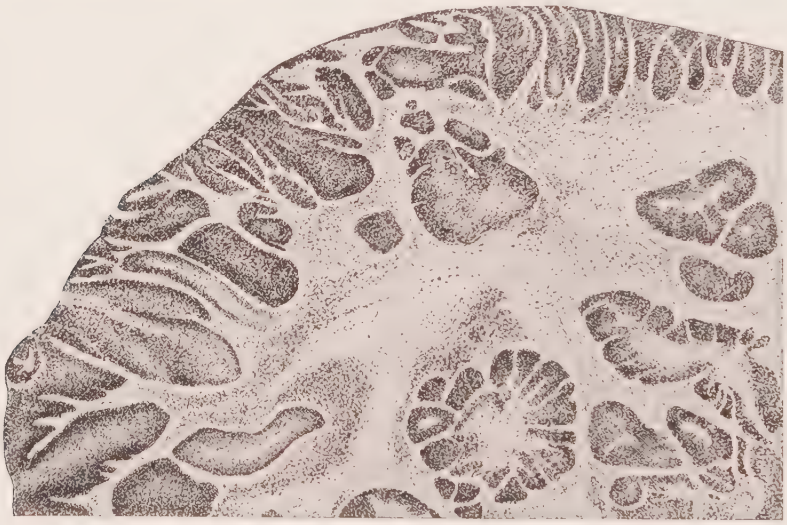


Fig. 849.—Non-cornifying squamous cell carcinoma of os uteri (basal cell); marked disintegration of centers of some nests; interstitial tissue infiltrated with eosinophiles. Nodule excised for examination from woman 54 years old. (Low power.)

hard keratosed papillary formations (see R. Meyer), or pointed condylomas, which are, however, always multiple, and also simple pedunculated papillary fibromas. They sometimes reach considerable size and may superficially resemble papillary carcinomas. Cervical carcinomas are histologically adenocarcinomas with marked tendency to form solid masses; less often they are squamous cell carcinomas derived from glandular epithelium after change into pavement epithelium, or from cylindrical epithelium, which during carcinomatous proliferation assumes the form of pavement epithelium, or directly from surface epithelium already undergoing metaplasia. In "deep cervical carcinomas" or "central cervical nodules" (lit. Benecke), derivation from rests of the Wolffian duct can be conjectured, (see p. 1464). Colloid carcinoma of the neck

(adenocarcinoma or squamous cell carcinoma) is rare (lit. by Miller, Meyer-Wirz).

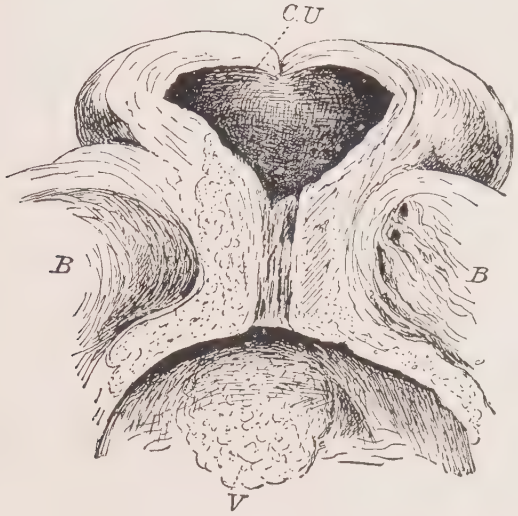


FIG. 850.—Infiltrating squamous cell carcinoma of os uteri with slight ulceration, stenosis of cervix and extension to bladder (B); CU, uterine cavity (pyometrium); V, carcinoma in vagina separated by macroscopically healthy tissue zone. Longitud. med. section: woman 67 years old; no infiltration of lymph nodes; ¾ nat. size.

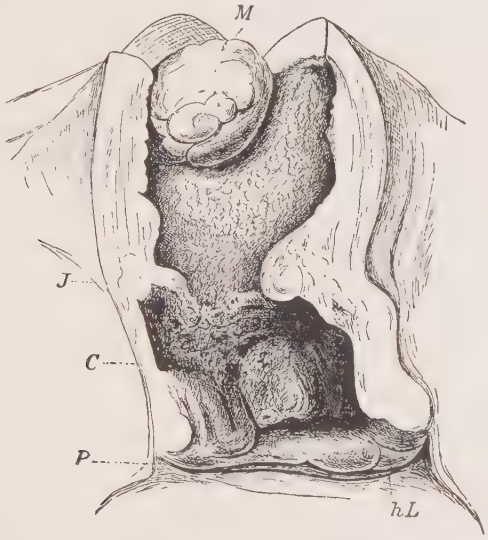


FIG. 851.—Cavitating carcinoma of cervix uteri (c) with tuberculosis of cervix and body. Internal os (J) macroscopically not, but microscopically transgressed by carcinoma; os uteri (P) preserved; posterior lip (hL); submucous myoma (M). There was an abscess in the space of Douglas, bordered by the patulous ends of the tubes which emptied 12 cm. above the anus as a fine fistula into the rectum; chronic cavernous phthisis of lungs, and tuberculosis of peritoneum, but no tuberculous ulcers; woman 50 years old; ¾ nat. size.

Mode of Extension of Carcinomas of the Neck of the Uterus.—Carcinoma of the os uteri soon pentrates the vaginal wall into the depths of

the portio and by way of the lymphatics into the submucous (parametral) tissue, while its extension to the cervical canal and the endometrium is later, although by no means rare; according to the investigations of Schottlander and Kermauner, it can be demonstrated microscopically in one-half of the cases of cervical carcinoma. The tumor tissues are white, granular and hard at first, then fatty, suppurating, softened, due to bacterial infection

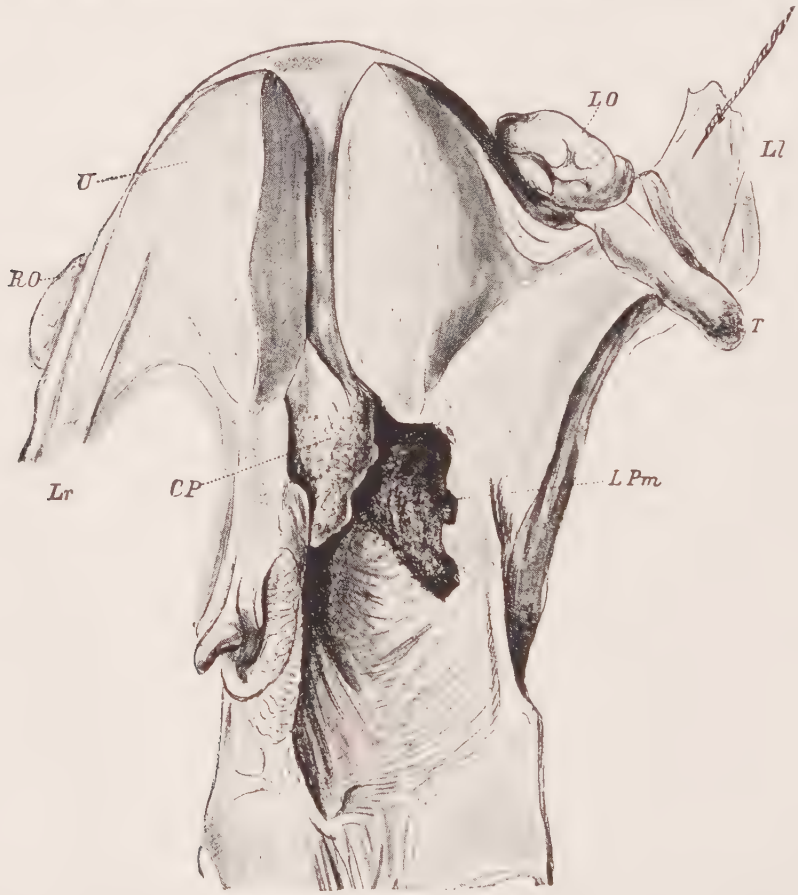


FIG. 852.—Ulcerative carcinoma of neck of uterus; remains of os uteri (CP); invasion into l. parametrium (LPm) rupture into vagina over the innominate line as far as the ileopsoas where there were abscesses and thrombosis of hypogastric and iliac veins; cicatricial carcinoma in pelvic cellular tissue; fundus much enlarged U, especially thick (so-called chronic metritis); LO, wrinkled l. ovary with tube (T) artificially pulled from behind forward; Lr, r. round lig.; Ll, l. lateral lig.; RO, right ovary; ¾ nat. size.

from without; a creamy substance can be expressed from them. As a rule, the carcinomatous areas disintegrate with suppuration, and frequently with extensive ulcerative loss of substance. They usually secrete a serous fluid which decomposes and leads to the hemorrhages which are frequently the first clinical symptom. The latter may even be dangerous

if large vessels are eroded, but are seldom directly fatal. The cervical canal with its surroundings appears as a denuded tube in the ulcerated vaginal vault (see fig. 853, p. 1678). But cases have been observed in which the infiltration of the os extended without macroscopic decay toward the vagina, bladder and in the cervical wall, causing pyometria by stenosis of the cervical canal (fig. 850). Cervical carcinoma, by its disintegration, causes cavernous hollows in the cervix and usually invades the parametrium promptly and deeply, as far as the pelvic bones, while the external os may be preserved and closed; more rarely it remains localized for a longer time in the hollowed cervix (fig. 851). If the destruction has progressed further, the starting point can usually not be determined, *i.e.*, whether from the os or from the cervical mucosa. It is then simply called cervical carcinoma; the whole cervical canal is destroyed and the parametrium massively infiltrated; the pelvic organs are glued in the pelvis, the ureters are compressed with its consequences, and there is excruciating pain. On the other hand, the parametria may open into the vagina as wide suppurating carcinomatous cavities which reach as far as the posterior bony pelvic wall. The rectum and bladder may open together as into a cloaca. The body of the uterus may be completely or partly preserved, and the carcinoma cavity is closed from the peritoneum in the median line. Adjacent organs, especially intestines, are adherent.

Carcinoma has developed in the uterine stump after supravaginal amputation of a myomatous uterus, but this does not mean that the scar is the starting point. Stump recurrences usually presuppose that the uterus removed supravaginally was carcinomatous (Fehim). In 1898 the author reported a case in which a carcinoma developed in a cervical stump left five years before after supravaginal amputation of a myomatous uterus (lit. by Winter, Burckhard, Uhl, Ekler, Heimann and others).

The uterine wall may be macroscopically free from carcinoma, or may be completely filled and thickened with white strands. Microscopically (see p. 1676), even in the very beginning of cervical carcinoma, strands of tumor tissue extend far up into the uterine wall, chiefly in the outer muscle layers, but, as stated on page 1676, in the mucosa also. Extension occurs by the lymphatics, first those which accompany the blood vessels, second, the intermuscular tracts (Seelig). Furthermore, the uterine endometrium may show multiple layers of surface epithelium, sometimes with pavement characteristics. Kraus considers the first suspicious, *i.e.*, a prestage of carcinoma; the latter has been described as a carcinoma which has crept upward from the cervix or has been implanted. Kraus (lit.), however, in cervical carcinoma with pyometritis, considers it a primary, multicentrically developed squamous cell carcinoma of the body of the uterus. Sometimes the uterine cavity is distended and filled with pus.

For pyometritis, see fig. 850. Infiltration of the parametrium is present in more than half of the cases (statistics by Koblank); invasion through the lymph channels (perivascular and perineural) is probably first arrested in the small interposed lymph nodules (comp. Kundrat). It leads to narrowing of the ureters, hydronephrosis (p. 1408), and pyelonephritis, which in turn frequently cause chronic uremia, to which is due the death of the majority of patients. The ureters may open directly into the

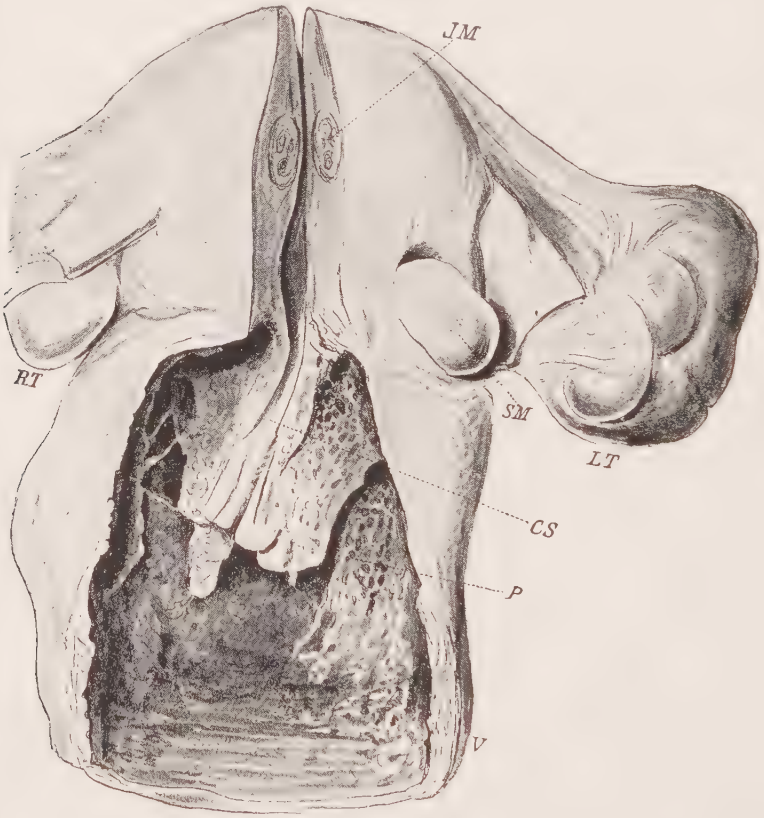


Fig. 853.—Ulcerative suppurative carcinoma of cervix with extensive invasion of vaginal vault. Left tube (LT) a pyosalpinx bound down by adhesions; RT, r. tube also dilated; external layers of cervix destroyed; denuded inner layers project into vaginal vault; CS, cervical mucosa; JM, intramural myoma; SM, subserous myoma; V, vagina; ¾ nat. size.

parametrial decomposition cavity. Infiltration of the parametrium after total extirpation, may lead to severe stenoses, *e.g.*, of the lower colon (comp. case p. 824). When the bladder wall or the rectum is invaded, broad fistulae appear between bladder, rectum and vagina, sigmoid or descending colon. The peritoneum over the parametrium above may be pushed upward; Douglas's cul de sac completely disappears. Adhesions, pseudomembranes, sometimes nodules, perhaps in large numbers, are

found in the peritoneum, or an extensive diffuse carcinomatosis. In a 58 year old woman the author saw a carcinoma the size of a hen's egg in a right inguinal hernia; herniotomy was done because of incarceration, but the occluding factor was the uterus larger than a child's head (carcinoma) and firmly embedded in the pelvis. Lymph nodes (lit. by Bruhns, Kroemer, Schottlander), *i.e.*, the hypogastric, sacral, iliac, lumbar, are infiltrated, (according to Wertheim and others, see Haendly, in 35%). For the cervix, the hypogastric and sacral nodes are the first stations (Peiser), but lymph nodes at a greater distance are sometimes involved (portal, bronchial, tracheal, supraclavicular, and others). Glandular and tubular epithelial inclusions in regional lymph nodes are of interest in the differential diagnosis, because they are also found if the primary tumor is of the squamous cell type. They develop by metamorphosis of endothelium into epithelial-like, sometimes ciliated, cells. These channels and tubes are sometimes quite similar to adenocarcinomas. Wertheim found these conditions, (described by others; Riess, Kroemer, Borst and others) in 13% of the cases of uterine carcinoma, and at first thought they were carcinoma. Others (Riess, Wulffing, Borst) thought they were evidence of misplaced and persistent rests of the Wolffian body. Finally R. Meyer found exactly similar formations in inflammatory inguinal nodes in cases free of carcinoma. (This was verified by Falkner, Brunet and others; lit. by Sitzenfrey, see H. Albrecht and Arzt.) The author has seen such hollow spaces lined with cylindrical epithelium in suppurating lymph nodes. Perhaps products of the metabolism of carcinoma exert an especially intensive irritation. It must be remembered, as Sitzenfrey points out, that ciliated retroperitoneal cysts may spring from these proliferations; according to Brunet, lymphangioendotheliomas, and perhaps even carcinomas, begin from them (Sick and R. Meyer, v. Gutfeld). Compare the work of Fromme in reference to reactive inflammatory changes in the lymph nodes in carcinoma of the cervix, which may lead occasionally to encapsulation and disintegration of tumor cells.

Distant metastases are said to be relatively rare, but according to the data collected at Basle, occur in ⅓ of the cases. Clinicians naturally consider them less frequent. Fritsch estimates them at only 3%. They are found most often in the liver and the lungs. An observation by the author in Basle was quite unusual (39 year old woman); a metastasis in the lower epiphysis of the femur showed extensive carcinomatous infiltration of the spindle shaped, swollen knee joint. Schiller mentions a case with involvement of both knee joints. In a 54 year old woman with primary carcinoma of the os uteri, Roedelius described secondary carcinoma of the calcaneum. The author has seen such a case (elderly woman). For secondary carcinoma of the ileum causing stenosis, see

fig. 393, p. 823. The author also found the thoracic duct involved. (Statistics in reference to metastasis of uterine carcinoma by Offergeld, Schottlander.) In the so-called inoculation metastases (Winter), there develop nodules from fragments of the tumor implanted into the tissues opened during operation (parametrium, vagina), or there develop apparently spontaneous nodules in the vagina and occasionally also at the urethral meatus. According to the opinion of Sanger, Veit, v. Herff and the author, however, these are probably metastases by the lymphatics,

Fig. 854.—Primary carcinoma of uterus from 65 year old nullipara; atypical hemorrhages and purulent discharge for 2 years, 14 years after menopause; adenocarcinoma, very poor gland imitations, many solid areas; surface epithelium transformed to epidermoid, (horny pearls); ¾ nat. size.

more rarely (partly retrograde) by the blood, or perhaps only an extension by continuity which is quite frequent (comp. under vagina).

Of the uterine carcinomas, that of the body is the least frequent (about 10 to 12%; according to Weibel, 5%). They usually grow from the gland epithelium, imitate the glands more or less completely (adenocarcinoma), or grow in solid cell masses (carcinoma simplex). More rarely it starts from the surface epithelium previously changed into pavement epithelium; these are usually cornified. But squamous cell carcinoma may develop from the gland epithelium, after preceding metaplasia. The tumor grows as single or multiple, nodular, broad based fungi, or villous growths, or sometimes as diffuse infiltrations. The author repeatedly saw them in conjunction with simple pedunculated benign polyps. The uterus is not of necessity much enlarged, but may reach the

size of a child's head. Its musculature becomes hypertrophic (comp. p. 1643).

In an adenocarcinoma of the body of the uterus (55 year old woman), the author saw the wall markedly thinned, the body slightly globular, the outside smooth, the inside fairly smoothly lined with caseous carcinomatous masses which infiltrated part of the wall. Macroscopically this was not distinguishable from tuberculous caseous pyometrium!

They appear preferably after the climacterium (comp. Weibel), and Deelman often found older women (nulliparas) the subjects. They grow relatively slowly, frequently produce regional metastases in the endometrium, and invade the musculature while disintegrating within. The outer muscle layers usually form bosses but preserve their architecture; later, they adhere to adjacent organs. The adnexa may escape completely, or be infiltrated at a late date. Operability is comparatively favorable and the results are relatively satisfactory. From the clinic of Wertheim, Weibel reports 51% complete cures. Metastases are rare, and mostly in the upper lumbar and inguinal glands (according to Weibel in 16%), the peritoneum, and ovaries. But occasionally, numerous metastases are observed in the various organs and in the bones. In a 73 year old woman, the author saw the uterus small, os and cervix free, carcinoma nodules and strands in vagina, infiltration of adnexa; no gynecological examination was made because the symptoms were only rheumatic pains in the region of the neck. Besides metastases to the liver, lungs, kidneys, thyroid gland and both thighs, diffuse carcinomatous infiltration of the spine was found.

The cervix, the os and the vagina are involved at a late date. The tumor grows downward by continuity and may simulate primary carcinoma of the neck, or produce obvious secondary nodules which occasionally seem as though they had leaped over quite healthy tissue (fig. 850).

Such foci are frequently called inoculation metastases (Hofmeier, Pfannenstiel). If careful examination proves conclusively that the entire tissue between the main tumor and the metastases is absolutely free of carcinomatous communications, and if a discontinuous deposit of cancer particles by the blood and lymph channels is excluded, then the secondary focus may be considered an inoculation metastasis; but as a rule they are generally blood or lymph borne, (comp. p. 1680, and Schottlander).

Carcinoma of the uterine endometrium may also be secondary to extension and spread of a cervical carcinoma.

Pfannenstiel believes it possible that a cervical carcinoma may be implanted on the mucosa of the fundus by instruments, and even spontane-

uos inoculation, *e.g.*, in pyometritis. Some gynecologists consider the simultaneous appearance of distant carcinoma nodules of cervix or os and the fundus as "double carcinomas." This term is not advisable, because it suggests two independent cancers, whereas one is revealed as the primary focus from which the other or others, which can be also observed in the vagina, are derived secondarily. The simultaneous appearance of a second primary carcinoma in the vagina (Hofer) is rare; it is more likely to occur in another internal organ, as the author, *e.g.*, saw in a 64 year old woman with carcinomas of the stomach and the os uteri, and at another time in a 74 year old woman with carcinomas of the gall bladder and os uteri.

Histology of the Carcinomas of the Uterine Mucosa.—In reference to their classification the following must be kept in mind: there is a temptation to differentiate them according to histological conceptions—from glandular epithelium and from surface epithelium. But to make such a distinction plausible in a given case requires that an early stage and the

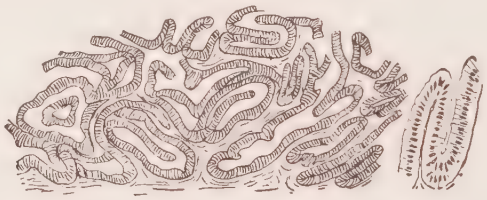


FIG. 855.—So-called malignant adenoma of endometrium; shape of gland labyrinth (after Schroeder); b, piece of epithelial layer of a so-called malignant adenoma (after C. Ruge). (Zeitschr. f. Geburtsh. and Gyn. Vol. 31.)

whole of the uterus be investigated. In practice, these conditions are usually not fulfilled. For these reasons, the author would follow the example of Schottlander-Kermauner (Obata, R. Meyer), and classify them formally as (1) adenocarcinomas, or primary glandular (partly secondarily solid), and (2) primary solid carcinomas.

1. One form of adeno- or primary glandular carcinomas (partly secondary solid) is that which is called, especially by gynecologists, "malignant adenoma" or "destructive adenoma." It is rare, and, according to the definition of E. Ruge, is a glandular carcinoma with determined cell formation and peculiar structure. Several types are described (Gebhard): the characteristic form is that in which a tangled skein of closely packed epithelial strands of one layer is seen (fig. 855), which has been compared to a labyrinth or a mass of earth worms. This form occurs in the uterus and rarely in the os where it starts from eroded glands. Another form (usually occurring in the cervix) shows acini close together, regularly and evenly arranged. But it is necessary to show that the acini form quite atypical shapes, possess no membrana propriae, and are closely

approximated. The relatively large nuclei lie in the centers of the uniform epithelial cells (fig. 855-b), which stain well, and are usually small cylindrical or cubical. Mirabeau speaks of one layer, but multiple rows, of cylindrical epithelium (see Klinger). The tumor is characterized further as a malignant epithelial neoplasm by its penetration of the mucosa, invasion of the muscularis and the production of metastases.

According to our conception, however, this is nothing but a cylindrical cell carcinoma with relatively perfect imitation of acini, *i.e.*, a very "ripe" adenocarcinoma (see Frankl, lit.). In reference to the os where the number of published cases is not large and can still be controlled, the author could show that such pure forms as correspond to the conception of "malignant adenoma" do not occur, or at least do not apply to the whole tumor. Indeed there always appear transitions to atypical structures, especially in the metastases, as far as it is a truly malignant tumor. On the other hand, it is by no means uncommon to find such adenocarcinomas characterized by polymorphous cells and their many layers in other organs (stomach, intestine), and that they show focal isolated areas corresponding perfectly to the single layer "acinar carcinoma of advanced maturity." Besides this, the same tumor often shows many transitions to solid cell nests irrationally called by some the only true carcinomas. Surely the carcinoma named adenocarcinoma, in spite of its imitation of acini, is also a true carcinoma! But while transitions from common adenocarcinoma to solid carcinoma may not be present, an adenocarcinoma nevertheless may develop fairly perfect acinar structure in one place, and less perfect structures in another; again, it may retain a more perfect form during its growth for a long time. These latter are the forms which cause the errors, and, as especially emphasized by the author (Virch. Arch., 154, 1898), produce the term "adenoma malignum," which is a practically useless designation. From other directions, as for instance, from Winter, the conception of malignant adenoma has been reduced to "adenocarcinoma," while Manauf Heurlin, on the other hand, would retain this designation, merely to name a histological form of carcinoma. Schottlander-Kermauner calls it "acinar carcinoma," "glandular carcinoma," or "high adenomatose" as a special form of adenocarcinoma (lit. in appendix). In the typical, common, less highly developed forms of adenocarcinoma, crude acini can be seen, *i.e.*, epithelial cells surround a lumen and some still show quite normal cylindrical shapes, while elsewhere the growth is characterized by multiple layers of polymorphous epithelium. But this architecture must not be mistaken for multiple rows (Thoma); the nuclei lie at various levels with a single layer of cells. There is also more marked variation in size and chromatin content of nuclei, also multiple nuclei, and the position of the plane of

mitotic figures is most atypical in direction (comp. Amann). Destruction or complete absence of basement membrane, the absence of boundary in their passage through muscle cells on their long axes, the development of large gyriform acinar units (see in the muscularis in fig. 857) or more marked lawlessness, the many communications between the acinar formations making it a veritable acinar labyrinth, all enter into the pictures (fig. 856). Much secretion by the cells as shown in fig. 856 is not common; in this they are similar to the microcystic adenocarcinomas of the intestinal

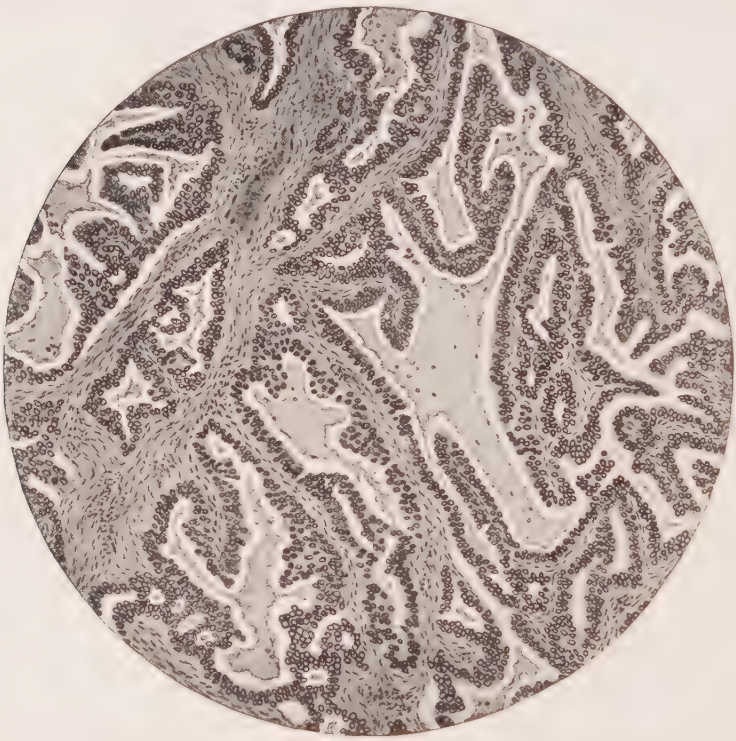


Fig. 856.—Adenocarcinoma (microcystic) of uterus; much mucous secretions in the gland imitations; women 41 years old. (Med. magn.)

tract. The lumena and the cells themselves contain mucus (comp. R. Meyer).

Finally, the diagnosis of carcinoma is made positive by the invasion of epithelial cells into the deeper layers of the wall (figs. 857 and 858), where they are frequently surrounded by reactive small cell infiltrations consisting of lymphoid cells, plasma cells and leukocytes. The appearance of numerous honey-combed, so-called pseudoxanthoma cells, is rare. In other cases, the acinar character is more and more obliterated; frequently but suggestions of acini are seen, and a tangled system of hollow spaces of varying size is penetrated by epithelial columns and ridges so that only

clefts and gaps remain (fig. 859). The epithelium surrounding the lumena is devoid of all regularity and does not form borders. Even solid epithelial masses and plugs are present (transition to solid carcinoma).

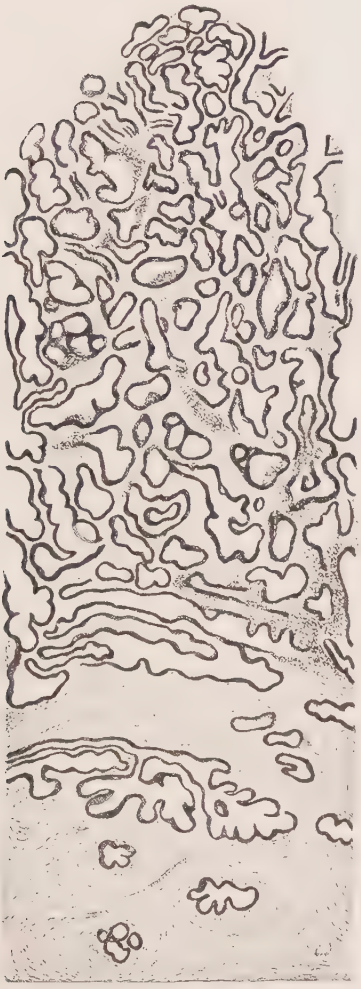


FIG. 857.—Adenocarcinoma of body of uterus; gland imitations fill mucosa thickly (S), also round cell infiltration; invasion of muscle. (Low power.)

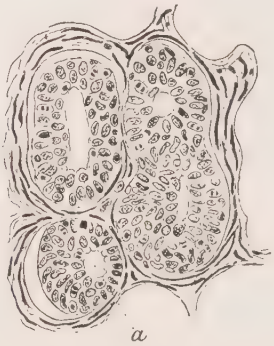


FIG. 858.—Adenocarcinoma of body of uterus; gland imitations in muscle; a, lumen; alveolar contents to the left partly detached from the wall from hardening process. (High power.)

FIG. 859.—Adenocarcinoma of body of uterus; very atypical gland imitations, some cut lengthwise. (Low power.)

Many adenocarcinomas have a villous papillary structure in certain areas (papillary adenocarcinoma), quite similar to villous vesical carcinomas (fig. 860). An irregular and thick epithelial covering of one or several layers, and more or less polymorphism covers the villous papillae, or lies directly on wide thin-walled blood vessels. Other areas may show the structure of a common adenocarcinoma. In these cases also, meta-

morphosis into pavement epithelium occurs (see for instance, Pees). In other cases, although focally there is still some similarity to acini, the bulk of the tumor has become a solid carcinoma, with the character of simplex—medullary—or a scirrhous tumor with polymorphous cells. There is much small cell infiltration in the immediate vicinity. In adenocarcinoma of the uterus also, there is sometimes a light, less often an extensive, metaplasia (according to Schridde prosoplasia, comp. R.Meyer) into cornified or cornifying squamous epithelium analogous to that in the cervix. It has been possible (as mentioned in the second edition of 1901) to demonstrate satisfactorily that the cylindrical cell masses differentiate into cornified squamous epithelium and epithelial pearls (see Frankel and Wiener

FIG. 860.—Papillary adenocarcinoma of uterus; polymorphous epithelium in several layers; ground work shows small cell infiltration. (Med. power.)

and Hitschmann, lit., Duse, Orvos, Buttner, H. Hauser, Loeb, who speak of adenocancroid, a term which the author ignores, disliking "cancroid" as a whole). Squamous cell masses may be so numerous that the tumor can properly be called squamous cell carcinoma. In other cases (see Emanuel), two histologically divergent forms may be found, which, however, are genetically uniform, and in which proliferating cells assume one or the other form. The cells of non-cornified metaplastic epithelium in adenocarcinoma are usually small, clear, rounded to polyhedral, sometimes with obliterated margins.

2. **Primary solid carcinomas** also show several variations. Most interesting are those in which the epithelium changes into stratified pavement epithelium, when it often, although not necessarily, is cornified and becomes the starting point of squamous cell carcinoma. But it must be mentioned that some authors (comp. for instance Herxheimer) deny the occurrence of metaplasia, and point out that the uterine cavity quite frequently contains "heterotopic" pavement epithelium; thus developmental variations (see p. 1625) must be taken into consideration in

reference to starting points. This is difficult to prove, and R. Meyer advises diagnostic caution when squamous cell nodules are found in hyperplastic acini of the endometrium and in carcinoma. An observation of Sitzenfrey, mentioned on p. 1667, is worthy of attention; multiple squamous cell carcinoma nodules developed in the acini of the endometrium, and also invaded the interstitial tissue. These relatively rare findings occur in older individuals in the majority of cases. Some of the plugs of epithelial cells cornify in their centers, and become epithelial pearls; others show no tendency to their formation and, as in fig. 861, the cells are small, stain strongly with eosin, possess a slightly staining nucleus, and show many fading margins. Both show early disintegration, but

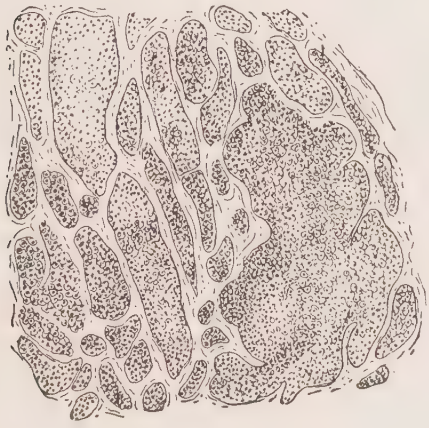


FIG. 861.

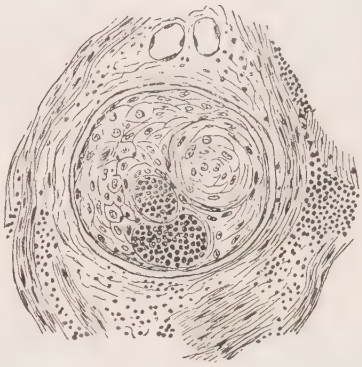


FIG. 862.

FIG. 861.—Squamous cell carcinoma of body of uterus (without epithelial pearls) derived from surface epithelium which had developed several layers; woman 52 years old. (Med. power.)

FIG. 862.—Cornifying carcinoma nest in distended lymph vessel of uterine wall; round cells in vicinity and within the nest surrounded by musculature; from the case below of both primary adenocarcinoma and cornifying carcinoma of uterus; woman of 64. (High power.)

penetrate the depths and proliferate through the tissue. Marked small cell infiltration is often found in the interstitial tissue (fig. 862), apparently independent of the carcinoma plugs. The surface of the carcinoma may be papillary or warty; the acini may disappear completely. A squamous cell carcinoma springing from the cervix may spread over the surface of the endometrium without "tumor" formation (Bencksier and others, see Lahm), a fact which Hitschmann finds hard to understand, but which Schauenstein considers not impossible, since some cervical carcinomas do extend as a flat coating over long stretches up the body. The mucosa disappears almost completely, and here and there, accompanied by marked small cell infiltration, the destructive invasion starts inward from the surface. There is, in this case, not merely an extension of carcinoma cells, but

the normal surface epithelium surrounding the tumor slowly undergoes carcinomatous change (Pronai). These pictures are known to the author, but require caution in their interpretation. The following is considered the criteria of carcinomatous change in the epithelial surface coating: no sharp demarcation of cells, especially in the basal layer, very irregularly clumped nuclei rich in chromatin, polymorphy of cells, absence of cylindrical basal cells with their pallisade-like arrangement, etc. But according to Kraus, it is possible to have the addition to the cervical carcinoma of a multicentric primary carcinoma of the fundus, *i.e.*, a second primary carcinoma (comp. below). The transformation of cylindrical epithelium into stratified pavement epithelium, which may cornify (ichthyosis, uterine psoriasis, see p. 1625), is considered suspicious, if not actually a prestage of cornifying carcinoma, (v. Rosthorn, C. Ruge). For this reason it deserves especial attention. Stratification and cornification, even the occasional formation of one or more epithelial pearls in the thickened surface epithelium or in an acinus (where of course metaplasia may be considered), are frequently found in a uterine adenocarcinoma. The author first observed the fact that the thickened metaplastic surface epithelium of the endometrium may simultaneously undergo malignant proliferation with a carcinoma springing from glands; thus this case had a double carcinoma, an adenocarcinoma of the body and a similarly situated cornifying squamous cell carcinoma. Both forms extended independently and the acinar imitations predominated. The squamous cell carcinoma above formed a thick layer with abundant epithelial pearls, but it also penetrated deep into the wall; fig. 862 represents a cornified carcinoma plug, filled and surrounded by round cells, and embedded in a lymph vessel of the musculature. In this case the adenocarcinoma also showed quite marked metaplasia into cornifying pavement epithelium, a change which occurs in other cases (comp. p. 1686). Hitschmann goes too far when he denies the possibility from principle; among his cases, there is not one which corresponds to the characteristics of the surface epithelium as described by the author (Hofmeier, Sitzenfrey). It also does not follow from the metaplasia demonstrated in the adenocarcinoma in our case, that the squamous cell epithelioma growing from the surface must be derived from the glands, as assumed by Eckardt and by Emanuel. The author has made it clear elsewhere (c. f. Sch. #7, 1906) that his primary conception of "double carcinoma" might be suitably modified. A pluricentric origin of carcinoma can be assumed—considering surface epithelium and acinar epithelium as at one level—whether the areas influenced by the cancer noxa have already partly or wholly undergone metaplastic changes or not, but since squamous cell and cylindrical cell carcinomas develop simultaneously, perhaps, later in their development,

the latter cells can, by metaplasia, assume the character of squamous cells. (Comp. Manu of Heurlin, Lahm, Strong and especially Keitler, exhaustive lit.)

Occasionally, calcium concretions (psammoma bodies) are present in abundance in degenerated cells of adenocarcinomas (Schmit), or in squamous cell carcinomas (Hitschmann), or are formed from secretions of noncornified squamous cell carcinomas (Kroemer), or from hyalinized tiny blood vessels or concentrically arranged connective tissue cells (Schutze). Such tumors are sometimes called psammoma carcinomas. In reference to the rare colloid carcinoma of the uterus, see Henkel and comp. p. 1674. Heuner reported a case of extensive hyalin degeneration of the carcinoma in which invasive proliferation of connective tissue into the degenerated masses caused many local auto-curative processes. See p. 1670 in reference to carcino-sarcoma.

See p. 1640 in reference to the combination of carcinoma and tuberculosis (lit. by Wallart). Concerning malignant chorioepitheliomas, see there; and for the rare carcinomas developed from adenomyomas, see p. 1667.

Metastatic carcinomas of the uterine mucosa form infiltrations or nodules; they are rare and mostly hematogenous in origin. Chiari observed them especially from mammary cancers. In a case of bronchial adenocarcinoma (58 year old woman), characterized by metastases to many organs,the author saw a flat carcinomatous infiltrate in the uterine mucosa. v. Franque reported retrograde injection of the lymphatics of the endometrium in tubal carcinomas; such retrograde invasion of the subserous and intermuscular lymph tracts by an ovarian carcinoma is frequent, (Romer, Glockner, Offergeld, lit., Arzt, Schmincke, Warstadt, Bassani and Zolewski in references to metastases in general). Metastatic carcinomas also occur in the myometrium. A metastatic carcinoma in Douglas's cul de sac (see p. 823) can infiltrate the uterus by way of the lymphatics; a tumor with lymph stasis produces a uterus of bony consistency (see Frankl).

Hints in Reference to Differential Diagnosis. Preparation of Curetted Material for Microscopic Examination.—Generally speaking, the tissue fragments removed by curettage in carcinoma are relatively hard, grumous, friable, cloudy, nontranslucent (opaque), white or whitish gray, sometimes very finely granular (alveolar); in sarcoma they are soft, like bloody brain substance, white or whitish red, floccular; in hyperplasia of the mucosa, they are membranous, easily crushed, like mucus, glassy, and transparent. In reference to the diagnosis of so-called malignant adenomas, see p. 1682. Good thin microscopic sections are essential. It is easy to recognize necrotic, suppurating detached fragments of myomas, or the remnants of

abortions, although beginners may mistake for carcinoma, islands of decidual tissue which lie between chorionic villi which have undergone hyalinization and loss of nuclei. Form changes of acinar cells due to post menstrual regeneration — enlargement, greater chromatin content, fusion of cells (formation of syncytium), the so-called atypical epithelial proliferations, the histological nature of which is most difficult to judge (comp. Benthin) — all must be taken carefully into consideration to avoid grave errors. It may happen that the curettage removes the carcinoma completely. The author saw such cases in which the extirpated uterus showed no trace of carcinoma (see Hess, v. Hansemann, Prym). Since macroscopic diagnosis is unreliable, it is essential to make a microscopical examination in all cases, if possible of every particle of the curetted material. The mass should be placed at once in fresh alcohol, or in 10% formalin solution for several hours. They may be sent away in these solutions to be examined microscopically, if the sections are made elsewhere. The fresh material including blood, may be placed in cloth and after washing away blood and mucus, the fragments can be examined. Tissue fragments which macroscopically appear sound must not be discarded; they may be the most important parts. At times, after unfruitful examination of many fragments, one will show characteristic carcinomatous areas, but they must be unmistakable large carcinomatous formations recognizable with low magnifications (see p. 1671). If the examination must be made quickly place the fragments in 95% alcohol and change often. It is advisable to keep the container in the incubator of 37°, or on the radiator to hasten fixation; then the tissue may be taken from alcohol and placed into ether-alcohol (equal parts) for some hours, when it is ready for the celloidin solutions, first thin, then gradually into thicker solutions (warming hastens the process). After they are poured into deep cups or cardboard boxes, and are dry to the hardness of cartilage, they may be cut with the microtome. Good sections can be procured in 12–14 hours provided the solutions and knives are good. It is best to use a stain which is familiar (haemalum-eosin or iron-hematoxylin with van Gieson stain are serviceable). There are quicker methods but they are less reliable. Some prefer embedding in paraffin. (Henke and Zeller's quick acetone paraffin method may supply useful sections within an hour, and are used by us in hurried cases; but they are less satisfactory.)

If the material for examination has been previously exposed to the x-rays, many degenerative changes in the cells must be taken into consideration; they are differentiated from the numerous spontaneous degenerative changes only by their greater extent (comp. Haendly).

See also Schottlander, histological diagnosis of tumors within the uterus.

Macroparasites of the Uterus.—Echinococci are usually submucous and may eventually be expelled spontaneously. Echinococci have also been observed in the adnexa, broad ligaments and the pelvic connective tissue.

Vagina

The vagina begins immediately behind the hymen and is about 7 or 8 cm. long. It is a fibro-muscular tube lined by a mucous membrane thrown up into rough warty folds (columnae rugarum). The membrane is built up of squamous epithelium, the lowest layers cylindrical just as in the epidermis. Glands are not present as a rule; the structures which resemble glands are, according to Eppinger, crypt-like epithelial depressions. Veith, however, saw "aberrant cervical glands" in the fornix of the vagina; and R. Meyer (lit.) found such structures in fetuses and the newborn, while Hohne described "heterotopic cervical glands." A number of authors believes there are glands, at least in certain individuals, which are lined by simple cylindrical epithelium (v. Preuschen and v. Herff, Widmer, lit., Pauliucu-Burla). The so-called vaginal "mucous membrane" is actually more of an epidermis. In the connective tissue which is rich in surface vessels, are small masses of lymphoid tissue.

The fluid which moistens the vagina comes from the uterus and from Bartholin's glands. The so-called "secretion" of the normal vagina is chiefly a powdery mass coming from exfoliated squamous epithelial cells. Normally it is strongly acid (Doederlein), and contains no pathogenic organisms, even in pregnant women (Bergholm); on the other hand, even in children, it contains in pure culture the bacillus described by Doederlein as a facultative anerobe producing lactic acid. Under pathological conditions it may contain many leucocytes, be weakly acid to neutral, thick yellow, greenish, foamy or watery. Under these circumstances, various pathogenic organisms, even anerobes, are present, and occasionally, the important streptococcus so common in puerperal fever. Pathological secretion occurs particularly in erosions of the os, in purulent cervical catarrh, in granular vaginitis, etc. (Doederlein). The normal secretion is bactericidal (self-cleansing of the vagina, Menge, Kronig), due according to Doederlein, to the lactic acid produced by the bacilli named after him; according to Menge, and Kronig, it is due either directly to the action of the anerobes living in the vagina, or indirectly to their metabolic products. According to Doederlein, streptococci are found only when the reaction of the vaginal secretion is neutral or alkaline. Conditions which tend to decrease the acidity (according to Loeser, those which interfere with the functional ability of its epithelium) such as menstruation, puerperium, foreign bodies, excessive coitus, and so on, influence the flora

of the vagina in such manner as to allow growth of pathogenic and saprophytic organisms (for further details, see under puerperal fever). Further details concerning vaginal mycosis on p. 1693. Schmidgall details the bacterial flora of newborn girls, and describes the slight bactericidal influence of the acid vaginal secretion on pathogenic organisms. Theoretically, the condition known as garrulitas vulvae, *i.e.*, the escape of gas bubbles from the vagina, is blamed on gas producing organisms (Veit). Bacteria are constantly present in the so-called gas cysts of the vagina (p. 1696).

The absorptive ability of the vagina is shown in various poisonings. The author saw fatal bichloride of mercury poisoning after douching in a woman who had had her uterus totally extirpated; further, he saw it in two cases in which sublimate pastilles had been placed in the vagina (see p. 1694). Haberda described arsenic poisoning from the vagina.

For malformations, see p. 1472 and following.

Inflammation of the Vagina. Vaginitis or Coplitis.—Various forms are seen. Acute colpitis is characterized by redness and swelling, wrinkling, and desquamation of the epithelial cells of the mucous membrane; such inflammations are called catarrhal, even though, in contradistinction to catarrhs of true mucous membranes, there is no mucoid secretion produced. The excretions or secretions consist of a serous, a milky or a purulent fluid, sometimes in enormous quantities, even in catarrhs (fluor albus).

If the epithelium is extensively desquamated, there appear red sandy erosive ulcers. Occasionally small vesicles appear in the epithelium (vesicular vaginitis).

Chronic catarrh is accompanied by a thick creamy or purulent secretion; the mucous membrane may become flat, gray or speckled with brown, or it may be swollen, reddened and infiltrated with connective tissue.

The overgrowth is seldom diffuse leading to thickening and wrinkling, but is, rather, circumscribed in papillary hypertrophies, somewhat similar to hard warts, and to those which occur in the vaginal portion of the cervix, or in softer branching outgrowths resembling pointed condylomata (see Kiesselbach). Numerous papillary hypertrophies give a raw, nutmeg grater-like appearance. Small outgrowths may be composed of lymphoid cells in the papillary and subpapillary tissues, quite similar to lymph follicles in appearance (granular or nodular colpitis). These granulations become erosive ulcers when the epithelium is desquamated. If they heal, or if the granulations disappear, brown gray spots, often in large numbers, are found as so-called Haller's stripes which give the vagina the appearance of a tiger's skin. A granular colpitis may, however, lead to adhesions, particularly in older individuals (vaginitis vetularum), by which areas

denuded of mucous membrane grow together; adhesions between the os and the fornix of the vagina are particularly common, either partly, so that individual bands are seen, or totally, so that the os, as in senile involution, can no longer be seen. Vaginitis exfoliativa is rare; it occurs at the time of menstruation and epithelial tags are expressed; it may occur coincident with an exfoliative endometritis (p. 1621).

Etiologically, varied causes may be mentioned: primarily it may be caused by the introduction of outside irritants of many kinds; thus too hot or too cold douches, foreign bodies, *e.g.*, tampons, ill-fitting pessaries, oxyuris vermicularis (see p. 854), gonorrhea, usually in children (see below). Secondary vaginal catarrhs are more common, following the irritation of fluids coming from a diseased uterus, or of the urine or feces in vaginal fistulae. Vaginitis may occur during the course of the acute infectious diseases such as measles, scarlet fever, small pox and others. Gonorrhea is a very common cause (see p. 1452). This latter type, however, does not occur because the gonococcus establishes itself primarily in the vagina, invading the mucous membrane and bringing forth a purulent inflammation; this is found only in the tender mucous membrane-like vagina of children as the important vulvo-vaginitis of infants or of young girls. This is very contagious and spreads in hospitals (even through fever thermometers and beds) as a wild epidemic (Sticker; see also Ense). These cases are in grave danger of metastasis to the tendon sheaths and to the joints, (usually monarticular, see Mattissohn, lit., Sutter, lit.). Gonorrheal proctitis is also common. In later years the vagina is spared from gonorrheal infection because of the density of its surface, and because of the normal bactericidal properties of its secretion (see p. 1691); even the purulent secretion discharged from the vagina contains no gonococci. The vaginitis present in these cases is more an effect of irritation or maceration from the gonococcic pus coming from the cervix or the urethra (see p. 1631). But beyond the youthful age, pregnancy and irritation from cervical secretion may in themselves be the causes of a blenorrhoic vaginitis (see Fritsch).

Pseudomembranous and Necrotizing Colpitis.—This condition may be a surface exudative process or a deeper necrosis (diphtheritic). Occasionally it is seen in conjunction with true pharyngeal diphtheria. In a child 1½ years old with gangrenous vulvo-vaginitis (noma genitalium), the author observed that the largest parts of both labia and hymen were completely destroyed, that the vagina was ulcerated and covered with a smeary exudate; diphtheria bacilli in pure culture were recovered from the vagina as well as from the pharynx. The infection had been primary in the latter place, but at the time of autopsy this was merely red and rough; the case was of decided interest because of the suspicion of criminal

destruction of the genitals. Freimuth and Petruschky in two cases of gangrenous vulvitis found diphtheria bacilli and brought about speedy recovery by the use of anti-diphtheritic serum. Cones described a similar case in a child eight months old; Leendertz observed post diphtheritic paralyses following the condition (bladder, rectum and other organs); see also v. Marschalko, concerning true skin diphtheria. But severe colpitis may occur in scarlet fever as well as in other acute infectious diseases of children, particularly small pox; it may take the form of innumerable ulcers in cholera, typhoid fever, and occasionally in dysentery (Klebs, Eppinger, Geipel). A pseudomembranous and gangrenous vaginitis and vulvitis are at times found in mercury poisoning (Bartsch, Orth, Wolffenstein, Hammer); the author has described two cases of this condition on p. 1692. Uremic ulcers are uncommon in the vagina; when present they are accompanied by a bloody, purulent discharge (Eichhorst). In other cases, the condition occurs as a so-called wound diphtheria in which a gray membrane, filled, as a rule, with streptococci, covers the surface; it is found in puerperal infections, or as a result of irritant discharges coming from tumors of the uterus or from decomposed urine in vesicovaginal fistulae, or, as the effect of an irritating pessary which often produces deep necroses. The condition is easily mistaken for thrush which shows large patches looking like sod. Pregnancy, old age, warm weather, favor the development of this fungus (see p. 560). More rarely, other fungi cause the condition, (monilia candia, leptothrix), see v. Herff. In the secretions coming from the cervix and the vagina in inflammation of the other genitalia, etc., Van de Velde found blastomycetes, but their pathogenic importance is doubtful.

In pseudomembranous colpitis the mucous membrane is swollen and corrugated and the surface of the rugae or the entire membrane is very red and covered with a light gray or green membrane. When the membrane is pulled off, deep ulcers remain. In rare cases, a deep necrotizing inflammation is quickly spread over a large part of the vagina causing gangrene; after healing, strictures or even complete atresia, are the results.

Phlegmonous perivaginitis is a suppuration of the perivaginal connective tissue which may spread far into the pelvic connective tissues (ascending perivaginitis). The entire passage may become gangrenous as its muscular tube is loosened over a long distance (dissecting phlegmonous perivaginitis). A phlegmonous perivaginitis may follow severe compression (*e.g.*, from obstetrical forceps), strong irritants (sesquichloride of iron, zinc chloride, Busse). According to Kretschmar, perivaginitis phlegmonosa dissecans is better called "idiopathic gangrene of the vagina" caused by the activities of an obligatory anerobic microorganism sui generis. The wall becomes necrotic, tissue is lost, there is

an evil smelling brown discharge, and the process spreads through the wall into the perivaginal connective tissue.

Simple Round Ulcers of the Vagina are Rare.—According to Zahn Beuttner and others, they follow primary changes in the vessels. The question, however, has not been completely settled, see Schroder and Kuhlmann. They are sharply outlined, rounded, with steep non-indurated edges and a red crater covered with pus. Other authors, including Veit, believe external influences and infections are necessary factors for their development and that diseases of the blood vessels and senile changes are of only predisposing moment. As a prestage of these ulcers, dark yellow, red rimmed necroses may be seen; the author found them in large numbers in an 87 year old woman.

Infectious Granulation Tumors of the Vagina. Tuberculosis.—Numerous disseminated nodules may be seen in children with uterine tuberculosis (see fig. 819). In severe infections of the bladder in children there occur many miliary nodules in the upper part of the vagina (the author). Adults occasionally develop lenticular ulcers, chiefly on the posterior wall near the uterus and usually accompanied by tuberculosis of the endometrium, more rarely of the vulva (lupus), of the urinary organs, or they are related to a recto-vaginal fistula. (Comprehensive lit. by Schroder and Kuhlmann.) Hematogenous miliary tuberculosis (from the lungs or bronchial lymph nodes), in which the vagina alone of the sexual organs is involved, may be seen in children, although rarely. The author has seen such cases (see also Springer); in a 15 year old girl he found many miliary tubercles in the vagina and a small caseous mass in the uterus above the internal os. (The tubes and peritoneum were without changes; the hymen was intact.)

Syphilis.—The primary lesion may occur at the introitus and spread to the vulva. This is less common than on the os. Hard chancres develop in this neighborhood. Ulcers may arise from papules. Gummas in tertiary lues or a gummatous colpitis in which the vagina is filled with friable gummatous masses (v. Winckel), are very rare and diagnosed with uncertainty, see Rille; the same is true of gummatous perivaginitis (Birch-Hirschfeld). Lit. by Schroder and Kuhlmann.

Cysts of the Vagina.—Larger cysts in the vaginal walls are rare and of varying genesis. There do occur rounded cysts to the size of a walnut, usually single, and found, as a rule, in the anterior and lateral walls. They are lined by epithelium, and contain a mucoid or serous water-clear or bloody pigmented fluid. They may arise from Muller's ducts (Freund), *i.e.*, they are rudimentary double vaginas and reach ordinarily not higher than the vaginal tissue; or they may be rests of the Wolffian (Gartner's) ducts (G. Veit) and usually extend higher than the former, perhaps even

into the neighborhood of the parovarium. In the diagnosis of cysts of the Wolffian ducts there must be taken into consideration the structure of the wall containing one to two layers of cylindrical epithelium, a connective tissue layer and a muscularis,—as well as its convoluted contour and its characteristic situation (G. Klein). Cysts of this duct may arise in any situation along its entire course from the parovarium to the body of the uterus; in the wall of the latter they occur laterally or laterally and anteriorly, or on the anterior vaginal wall, and in this situation, above more laterally, and below more anteriorly (Dohrn, Rieder, Kuster). The parovarian cysts (p. 1569) are exactly similar to these vaginal cysts. (*c*) Larger cysts may result from dilatation of lymph vessels, from traumatic lymph extravasations (Thorn), from paravaginal hemorrhages (Gotthard), occasionally from echinococci. (*d*) Cysts may also arise from the vaginal glands (see p. 1691).

In the so-called cystic colpo-hyperplasia, (v. Winckel), the surface contains many small flat cysts containing gas, and therefore called gas or air cysts (Schroder), or emphysematous vaginitis (Zweifel and others). They are said to be lined by epithelium, and, according to v. Winckel, contain the gases of decomposition (trimethylamin) arising between adherent portions of the mucous membrane. According to Zweifel, however, they originate in vaginal glands (see p. 1691). Others found no epithelium in these gas cysts, and Chiari unquestionably showed that they come from lymph vessels, the endothelium of which develops into many giant cells. (The author can verify these findings.) According to Eisenlohr, G. Klein and others, the formation of gas is due to anerobic gas producing bacilli (bacillus emphysematous vaginae). The softening and hyperemia of the vagina during pregnancy seem to provide a good culture medium for these bacilli: Lindenthal produced similar pictures in pregnant animals by introducing pure cultures of these organisms. Lit. Eug. Fraenkel, Nowicki, Roman, also Aichel; (compare cystic-pneumotosis of the intestines, p. 767).

Tumors of the Vagina

Papillary fibro-epitheliomas (pointed condylomas) were described on p. 1692. Fibromas (lit. Kehrer), or fibromyxomas, are rare and usually without significance. Schmauch described a unique plexiform neuroma. Myomas (leiomyomas), or the more frequent fibromyomas, particularly on the anterior wall, are comparatively rare, but occur in every age and may reach the size of a child's head; some of these tumors are adenomyomas (see p. 1662). Rhabdomyomas and rhabdomyosarcomas are rare. Sarcomas, among them melanosarcomas, (Fraefe), are quite uncommon (lit. in appendix).

In children, polypoid, grape-like papillary myxomas, myxosarcomas or edematous sarcomas occur; these are always congenital and are quite similar in appearance to the grape-like sarcomas of the cervix in adults. These tumors prefer the anterior vaginal wall and may extend into the bladder. The collection in Breslau contains such a preparation obtained from a 2½ year old child (Soltmann). In a 3 year old child, the author saw a basket-like polypoid myxoma of the posterior vaginal wall almost as large as a chestnut. It contained many plasma cells. They may contain cartilage and cross-striated muscle fibers, in which case they are plainly mixed tumors, and their complicated structure leads to the assumption that they are developed from embryonal inclusions (see p. 1634). Wilms is of the opinion that all the tumors containing cross-striated muscle fibers arise from the primitive vertebrae, or the myotomes, and that those in the vagina, as well as those in the cervix, arise from germinal cells dislocated by the caudal growth of the Wolffian ducts into the genital sphere; see also R. Meyer. Round cell, polymorphous cell, giant cell, angio-cell sarcomas also occur, as well as pigment sarcomas. In adults, sarcomas behave differently; some diffusely infiltrate the vagina, others develop enormous nodules. Spindle cell and alveolar sarcomas are also encountered. It is necessary to avoid mistaking richly vascular sarcomas for primary chorion-epitheliomas of the vagina (see p. 1698). Vaginal sarcomas are sometimes secondary to primary uterine tumors (p. 1669).

Carcinoma.—Secondary carcinomas are quite common, from the os or from the rectum (fig. 853), but they seldom come from carcinoma of the vulva.

Discontinuous carcinoma nodules occur in the vagina from uterine carcinomas (fig. 853). Many are of the opinion that these are implantation metastases (see pp. 1680, 1681 and 1707, Fischer and others), but possibly more attention should be paid to retrograde transport through the lymph and blood channels. The author has been able to demonstrate these paths in an accurately studied case, and has seen many similar instances; Milner (lit.) doubts the occurrence of implantation carcinomas in the vagina.

Primary carcinomas, usually cornifying squamous cell varieties, are comparatively rare; they may occur as cauliflower-like nodules, as ulcers, or as diffuse infiltrations, usually on the posterior upper wall.

Adenocarcinomas likewise are seldom found; their origin is either from the disputed Gartner's ducts, or, as Hohne (lit.) showed, from heterotopic cervical glands (see p. 1691); (Hirsch).

The spread of adenocarcinomas to the vulva is common, but to the uterus is uncommon; they often invade the bladder or rectum and form fistulae. Their growth is usually quite rapid, according to Williams, about

1½ years. The inguinal nodes and the upper ⅔ of the rectovesical nodes are first involved (Kronig).

Etiologically, those carcinomas, usually of diffuse infiltration (v. Franque), which arise in a previous leukoplakia (Reclus, Lohnberg, lit.) are interesting; according to v. Franque, leukoplakia of the vagina practically always becomes carcinoma in the end. In one case, Edelberg held responsible the pressure of a pessary which had been in the vagina for twelve years. Landau described a carcinoma in a forty-eight year old woman which arose in the scar of an operation done twenty-four years previously. For carcinoma of the vagina in prolapsus, see Kleemann (lit.). In the differential diagnosis there must be considered: tuberculosis, soft chancres which are often multiple gummas, and less frequently, hard chancres.

Metastases in the vagina are quite common in malignant chorion-epitheliomas of the uterus (p. 1744), but they are rare in the tube (Vassmer); in the former it is believed that the path is by way of retrograde embolism (Pick). Usually they are brown-red, rounded tumors. Furthermore, malignant chorion-epitheliomas grow in the vagina in cases in which the uterus contains a malignant hydatidiform mole (Brenner, lit.), or even when the hydatidiform mole shows no malignant criteria (Pick, Neumann, Poten-Vassmer, Schickele). But primary (ectopic) chorion-epitheliomas of the vagina are to be found usually when there is a question about the normal condition of the placenta in the uterus, or after hydatidiform mole without the formation of tumors in the uterus or in the tubes. These usually begin in the first month after childbirth and very rarely during the period of pregnancy; see Schmorl, Hubl (lit.), Schmauch, Marullaz, Risel (who observed tumors in the lungs and liver), Hicks and others; see p. 1744, where the case of Walthard is described; the latter showed that the placenta was entirely intact. After operative removal of the vaginal tumor, the uterus may remain healthy even though it contain a hydatidiform mole. But in these cases, it is necessary to show, by repeated currettage, that the uterus remains healthy. The criteria which are useful for the microscopic diagnosis will be found on p. 1745.

Parasites of the Vagina.- Trichomonas vaginalis (see p. 857) is common in the vagina and is probably of some clinical interest (see, among others, Seitz); they are found in the trichomonas colpitis or vaginitis simplex, as it has been called, and according to Schroder and Goerbig, are possibly associated with ovarian insufficiency inasmuch as the chemistry and the flora of the vagina are disturbed in this latter condition. Thrush is quite common, especially in pregnant women (see p. 560). Oxyuris vermicularis (see p. 854) may reach the vagina from the rectum.

Changes in Position.—Inversion of the vagina, *i.e.*, turning inside out of the anterior or the posterior wall into the lumen, is quite common in the relaxations of improper involution during the puerperium or prolapsus of the uterus. If the inverted wall appears in front of the vulva, the condition is known as prolapsus of the vagina. The protrusion is often annular and total, and probably a part of the picture is a consequence of retroflexion of the uterus. Partial prolapse is observed in partial perineal defects. The posterior wall of the bladder (cystocele) and the anterior wall of the rectum (rectocele) may bulge out and downward (see p. 1614). Dilatation of the bladder and of the rectum are occasional causes of inversion. More rarely it is due to ovarian tumors (ovariocele vaginalis), loops of intestine (enterocele vaginalis), or exudate into Douglas's cul de sac (hydro- or pyocolpocele), etc.

Traumatic and Spontaneous Solutions of Continuity. Foreign Bodies.—Injuries during childbirth are quite common. Crushes and tears occur in spontaneous or instrumental deliveries. Apart from hemorrhage, the most serious consequences are puerperal infection, the development of permanent tears, or abnormal communications, *i.e.*, fistulae, between the vagina and the neighboring organs. Lacerations occur most commonly from overstretching by too rapid passage of the head, and appear as tears of the perineum (p. 1701) or of the os uteri. As a rule, they are longitudinal in the posterior wall, less often, oblique, penetrating into the peritoneal cavity through the posterior vaginal tissue. Rectovaginal fistulae are quite common. Instrumental delivery, and especially cranioclasty, may cause severe damage (Kaltenbach). Pressure exerted by the child on the anterior and posterior soft parts caught between it and the symphysis leads to bladder fistulae and occasionally to vagino urethral fistulae.

Rupture of a vessel during childbirth may cause so-called hematoma and thrombosis of the vagina and vulva; the hemorrhage is into the perivaginal connective tissue, behind the labia. It may follow instrumental crushing or rupture, or direct tears by overstretching in the second stage of labor. The presence of varices, quite common during pregnancy, is a predisposing cause. The mass of blood is sometimes as large as a child's head, and death from hemorrhage may result from its escape, either primarily or after necrosis of the coverings. Or, the hematoma may soften and be gradually reabsorbed. In a 29 year old woman, the hematoma was the size of a small fist; it had come from a ruptured varix in the uterovaginal plexus; the soft blood clots pushed the posterior vaginal wall hemispherically into the lumen and burst through an opening admitting two fingers; profuse bleeding combined with air embolism caused death. Lit. by Reich, Wimpfheimer, lit., Beckmann. For varicose ulcers of the vagina, see Schroder and Kuhlmann.

In addition to the injuries during childbirth, those arising from other causes may be considered. Foreign bodies, particularly pessaries, may, with long continued use, lead to pressure necrosis and thus to perforation of the walls. This is particularly true of rounded pessaries, (the author even saw "bitter-apples" used for this purpose in Silesia!) and very grave consequences may follow: the entire wall becomes necrotic, and the picture is quite similar to the decubitus in the rectum in coprostasis. Other foreign bodies introduced into the vagina (see lit. by J. Veit, Hofmeier) lead to severe injuries to the bladder and the rectum. Spontaneous solutions of continuity are most frequent in connection with malignant tumors, particularly carcinomas, but they also occur, though more rarely, in gangrenous inflammations. Fistulae to the bladder, the rectum, the urethra are quite common.

Vulva

Anatomy.—The vulva consists of two concentric annular folds, the larger and smaller labia; between them is the rima pudendi with the openings of the urethra and the vagina. The labia majora are united by the frenulum labiorum, behind which is the fossa navicularis, a deepening of the vulvar orifice. Superiorly, the labia majora blend with the mons veneris. This and the neighboring skin are covered with hair after the onset of puberty. The labia minora or nymphae are hairless; they arise on each side of the perineum and extend anteriorly to the clitoris, which hangs downward from the pubic arch. They then divide into two small folds, one of which is inserted on the under surface of the glans clitoris, the frenulum clitoridis; the other rises above the glans to meet with the corresponding fold of the other side and form the prepuce of the clitoris. The space between the inner surface of the nymphae from the urethral opening down to the opening of the vagina, is called the vestibule. Under the mucous membrane of the vestibule are venous plexuses which enter the corpora cavernosa of the clitoris. The inner surface of the nymphae and the vestibule no longer have the character of the outer skin, and are very richly supplied with large sebaceous glands. The mucous membrane contains papillae and is covered by many layers of squamous epithelial cells. Concerning glands of the clitoris, see Boyd.

The **hymen,** (see p. 1464), is a richly vascular half moon-shaped reduplication of the mucous membrane. If this structure is torn by coitus or other force (defloration), there remain, after healing, small nodules known as the myrtiform caruncles. The hymen fimbriatus, one of the several varieties of the structure, is, (without defloration), notched or fringed by deep grooves, a fact of medico-legal importance.

Circulatory Disturbances and Injuries.—**Hyperemia** as an active process follows various forms of irritation (in inflammations); as a passive process it is seen in the general circulatory disturbances of heart and kidney diseases, or as a result of local stasis in the pelvis. If the obstruction reaches a high degree, there appears an edema in which the soft watery labia majora become like large sacs. As a rule, the vulva is edematous in pregnant women and during the puerperium.

Inflammatory edema occurs in the neighborhood of ulcerations, infected tears of the vulva, (the so-called puerperal ulcers), in deep inflammations spreading from the soft parts of the pelvic bones, etc. Varices are frequent usually from the pressure of a pregnant uterus. They may rupture and cause fatal hemorrhage.

Hemorrhages.—External hemorrhages from the surface occur from varices, ulcerations, traumas, rape, etc., and are particularly common after childbirth because of the swelling and hyperemia of the tissues; they may even be fatal; some hemorrhages enter the cellular tissue of the labia majora producing hematomas and thrombus vulvae (see hematoma vaginae, p. 1699).

Injuries.—If small tears of the vulva during childbirth become infected, they produce the so-called puerperal ulcers.

Perineal Tears.—When the child passes through the vagina and vulva too rapidly, or when the size of the head is too large, the perineum ruptures. Most tears involve the vagina, also, generally its posterior or lateral walls. There are differentiated: surface or incomplete, and deep or complete tears. In the former, the tear includes the skin as well as the constrictor vaginae and the transverse perineal muscles. It may begin at the posterior commissure, or in the vagina. Complete tears pass through the sphincter ani more or less deeply into the rectovaginal septum, in rarer cases, even into the rectum, but more often lateral to this structure. Exceptionally, the frenulum and the anus are not torn as the tear passes through the middle of the perineum, and even the child may pass through this vagino-perineal fistula (central perineal tear).

Inflammations.—We differentiate the inflammations of the skin surfaces and of the mucous membrane. The former are the various skin diseases such as erythema, eczema, herpes, acne, furuncles, etc., (see under skin); the latter constitute true vulvitis.

Acute vulvitis (catarrhalis) is characterized by swelling, softening, redness, and occasionally, purulent secretion. It arises primarily from masturbation, mechanical irritation during walking, uncleanliness, but particularly from gonorrhea, in adults as well as in children. In the latter, the primary vulvovaginitis often spreads to the rectum and the urethra, but hardly ever to the bladder, (Flugel); in adults, however, it

may be a secondary effect of the pus poured from the uterus, bladder, Bartholin's glands, etc., (see gonorrhea, p. 1452). A secondary vulvitis may occur from other catarrhs of that vicinity, *e.g.*, from the discharges of decomposing tumors of the uterus or the vagina. Chronic vulvitis takes the form of diffuse hypertrophy or overgrowth of the papillary bodies, papillary hypertrophy, with transformation into pointed condylomata, and leucoplakia (Bonney). Leucoplakia prefers the outer surfaces of the labia majora and often spreads to the skin of the thighs and perineum. The principal, and ofttimes the only clinical symptom, is pruritus. Histologically, there is diffuse thickening of the epithelium with growth into the deeper tissues; lymphocytic infiltration is found in the underlying thickened parts which are also traversed by connective tissue fibers; the elastic fibers in the upper layers, disappear. Carcinomas, perhaps even multiple primary growths, frequently arise from leucoplakia, (see the case of Fromme following pruritus of 6 month's duration). The histological details may be found described by Bucura. In kraurosis vulvae, (Breisky), (after a previous inflammatory hypertrophic stage?) there is disappearance of the tissue with shrinkage; this may reach such marked degree that the labia majora become flattened and the labia minora and the clitoris disappear entirely. The region of the urethral opening as well as the vagina may be coinvolved. The vaginal orifice is narrowed. The mucous membrane becomes hard, pale, reddish gray, or spotted white; it is dry, glistening, infiltrated by lymphocytes and plasma cells, and contains ectatic blood vessels here and there; following tears, frequent because of the hardness of the tissue, there develop eczema and small ulcerations. In the deeper layers of the tissue, sebaceous glands, fat, vessels, connective tissue and nerves disappear, while, in the upper parts, there are hyperplastic processes leading particularly to hyperkeratosis (Heller). But these findings are not constant (Darger), and newer investigations by Berkeley and Bonney, which allow of a sharper differentiation between kraurosis and leucoplakia (see also Jayle), show in the former, particularly in the red spots, an abnormally thin epithelium. A. Martin believes that kraurotic tissue may be the basis of carcinoma, a wide-spread viewpoint, while Berkeley and Bonney believe it has nothing to do with carcinoma, but that leucoplakia is a frequent forerunner of this tumor; they saw carcinoma of the vulva in their collected cases of leucoplakia. As causes of kraurosis many, perhaps chemical, irritants are important (diabetes, etc., Jung, v. Linck, lit.); Gardlund (lit.) believes the cause is an endogenous constitutional affair, not infrequently a neurosis. Jayle believes disappearance of ovarian function following castration or atrophy is an important factor. **Pseudo-membranous and diphtheritic necrotizing inflammations** may occur in puerperal injuries or following

pharyngeal diphtheria by the carrying of infection through a finger. See under diphtheritic vulvovaginitis, p. 1694.

Gangrenous inflammations occur in the severe infectious diseases, but are particularly prone to follow lesions in the puerperium. In other cases, they are part of the specific ulcerative soft chancre when it becomes a phagedenic ulcer; this may be very destructive. Infection by the diphtheria bacillus is known to lead to gangrenous vulvitis or noma of the vulva (see. p. 1693). The rare gonorrheal ulcer is characterized by a spreading serpiginous process in the subcutaneous tissue with undermining and destruction of the skin (Thalmann).

Descending infection, particularly in the labia majora, may follow phlegmons in the pelvic organs or puerperal ulcers. In severe typhoid fever, ulcers of the vulva and vagina have been found which yielded pure cultures of typhoid organisms (Lartigau, Scherber).

The acute ulcer of the vulva may be mistaken for tuberculosis because of its tough, sharply outlined, undermined edges and its purulent necrotic floor (pseudo-tuberculous ulcerative vulvitis, Sachs); it is caused by gram positive bacilli (Lipschutz), which Scherber (lit.) has identified as the bacillus of Doederlein. Scherber, therefore, speaks of them as "vaginal bacillus ulcers."

Infectious Granulation Tumors of the Vulva. Syphilis.—There occur initial scleroses and hard chancres, as well as primary and secondary papules; the vestibule, the nymphae and the commissures are preferred. For elephantiasis, see below. See under skin, concerning venereal but not syphilitic, soft ulcers with thick rims and moth-eaten floors covered by whitish yellow pus.

Tuberculosis (lupus) is quite uncommon. The ulcerative forms destroy considerable tissue; the hypertrophic forms, on the other hand, produce tumors as large as pigeon's eggs (lit. Bender). Lupus may be confused with luetic processes and with beginning carcinoma; it may be followed by elephantiasis vulvae, (Jesionek). Tuberculosis in this region is probably always secondary, but this cannot be demonstrated clinically (see Kroemer, Seiffert). Chronic tuberculous ulcers of the vulva are discussed on p. 1705.

Actinomycosis has been described as a primary condition in the labia (Bongartz). The term plasmocytoma, (see p. 272), has been used by L. Martinotti, to describe a small tumor on the vulva consisting of practically nothing but plasma cells.

Hyperplasias and Tumors of the Vulva.—In elephantiasis the cutaneous and subcutaneous tissues become much thickened; the pipillary bodies and the epidermis may or may not, be involved in the hypertrophy. The affection may include the entire vulva, or, if only a part, principally the

labia majora and the clitoris. Abnormal enlargement of the labia minora is called the Hottentot's apron. Growths weighing twenty pounds and reaching to the knees have been described. The enlarged parts are made up of tough, bacony, or soft edematous connective tissue; the outer skin cannot be separated from the underlying tissue masses. There develop flat hard, or soft diffuse thickenings, or roughly nodular growths, often with pendulant moluscum forms with fairly smooth or wrinkled or papillary surfaces. The papillae are thick or thin; if the latter, the growths are cauliflower-like, similar to colossal pointed condylomata, but more frequently they are harder and paler than these (white condylomas). Mechanical or decubital ulcers may appear on the surface, since the nutrition of the parts rests on such insecure ground; they are likely to become chronic. Lymphorrhagic forms occur. When much fatty tissue appears in the deeper layers, pendulous lipomas appear (lit. in appendix).

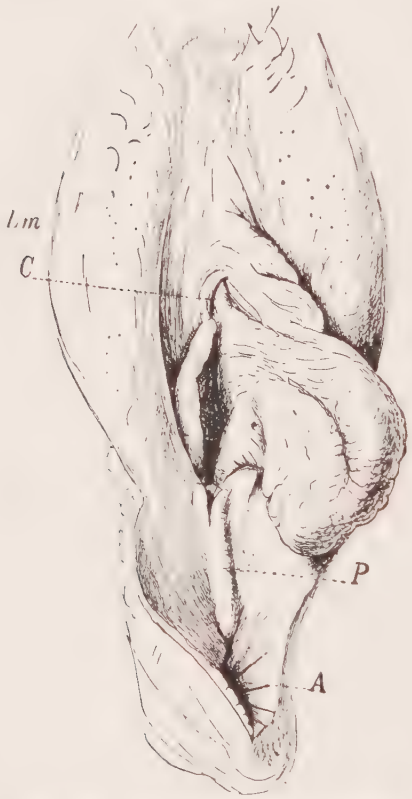


FIG. 863.—Elephantiasis of left nympha; C, clitoris; L. min. s. Labium min; P, perineal raphe; A, anus; L, labium major; $\frac{3}{4}$ nat. size.

Etiologically, there may be considered: (*a*) congenital anlage, *e.g.*, in the form of congenital lymphangiectasias, or cystic lymphangiomas in the tissues in which an elephantiasis arises. (*b*) Acquired elephantiasis is more common, due to all sorts of conditions which lead to lymph stasis, *e.g.*, acute inflammations (erysipelas), and chronic inflammations in which lymph vessels and lymph nodes are destroyed or obstructed, specific processes, tumors, such as carcinoma of the rectum (Muller), trauma, etc.; syphilis also is said to be a cause (Bamberg, lit.), although, according to Matzenauer, no syphilitic criteria can be found in the condition. In tuberculosis, the elephantic sclerosis is due to obstruction of lymph channels; see Forgue and Massabuau, Daniel, (lit.). Masturbation is said to be a cause. When the condition is accompanied by ulceration, as a consequence of extirpation of the inguinal lymph nodes with their resulting edema, growth of connective tissue and destruction of normal tissues, F.

Koch calls it "ulcer of the vulva," analogous to chronic leg ulcer and to ulcerating stricture-producing processes in the rectum (see p. 790). Bjorling speaks of the condition as "vulvo-anal elephantic ulcers" and sharply differentiates them from all specific ulcerative processes. Linner (lit.) calls them chronic simple ulcers. Recently, the tuberculous nature of the ulcer has been emphasized (Rutter, Schade, lit.). The term "rodent ulcer of the vulva" (Rutter) should not be used, since the pathology of a rodent ulcer is that of carcinoma (see under skin). By many, the ulcers are also called "esthiomene" (meaning devouring).

Pointed condylomata (condylomata acuminata) are frequent in women who have borne many children, and also in gonorrhea; they are acutely

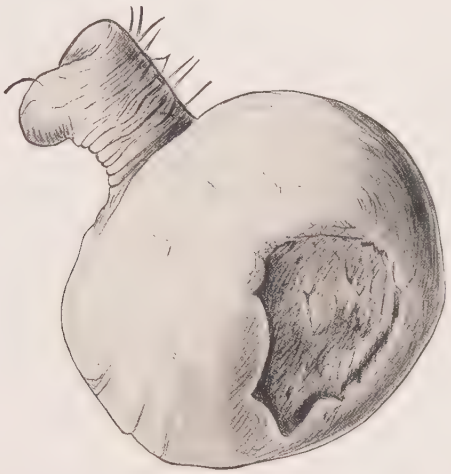


FIG. 864.—Pedunculated fibroma of r. labium major; deep decubital ulceration; woman 48 years old; ¾ nat. size.

appearing inflammatory papillomas (papillary fibroepitheliomas), with richly vascular papillae covered by many layers of epithelial cells. They may reach the size of an apple, and are composed of innumerable cornified papillae. Cases have been observed in which a condyloma became carcinomatous or recurred as a carcinoma. The author studied such a case in a 37 year old woman; following gonorrhea there developed many pointed condylomata in the anal folds; these were removed. A year later there was a papillary squamous epithelial carcinoma removed from the same place. This returned shortly in the form of an ulcer as large as a soup plate. Death occurred in the same year. Metastases in the clitoris. Microscopic examination was made at every stage.

Fibromas, fibromyxomas, fibromyomas, usually arise in the labia majora, from the round ligament or from the clitoris. Circumscribed, hard, nodular, or polypoid tumors may become pedunculated, pear-shaped

and reach the size of a child's head. The outer skin surrounding the tumor may be raw, smooth, papillary, or ulcerated (fig. 864). Lipomas may superficially resemble fibromas; they also may become pedunculated. Angiomas, chondromas, and neuromas are very rare. Pick and Gross (lit.) have described a particular species of adenomas, nodular multiple "hidradenomas," similar to a sweat gland, composed in part of simple glandular and in part of intracanalicular structure. The author has seen these tumors in a number of cases; thus, in a 51 year old woman the tumor was as large as a pea and lay completely isolated in the corium; the glandular formation showed two layers of epithelial cells like those in sweat glands, limited by an elastic membrane (fig. 1055). See Schroder also. They may be mistaken for metastases of adenocarcinomas! But metaplasia into adenocarcinoma does take place (Frattin). Schiffmann described a so-called adenocancroid of the sweat glands of the vulva. Retention of smegma in the clitoris may produce a tumor-like hardness.

Malignant Tumors.—Sarcoma of the vulva and particularly of the clitoris is rare. Among others, there occur spindle cell and pigment sarcomas which are said to be influenced by pregnancy (Labhardt). Carcinomas are more common and arise from the squamous epithelium of the surface (relatively frequently from leucoplakia, see p. 1698) or if adenocarcinomas, from Bartholin's glands, sweat glands, from the urethra or from the hymen. See Tobler concerning gelatinous cylindrical cell carcinoma. These are most commonly seen in the clitoris and in the folds of the nymphae, but they may be found even on the hymen (Frankl); they give rise to soft tumors, extensive ulcers, or papillary or nodular mollusc-like growths. (Lit. in appendix.) The author saw a broad based pedunculated squamous epithelial carcinoma of the clitoris in a 25 year old woman.

Carcinomas are apt to be particularly malignant in this situation (Kustner); they quickly involve the neighboring nodes and local recurrences are rapid. But on the other hand, the author has seen cases in which the duration of the disease, in spite of many recurrences, was six years; in one case it was more than sixteen years (see Keller); in the former, in a 26 year old woman, the author saw the third recurrence (neighboring lymph nodes) six years after a carcinoma of the vulva was removed and four years after the carcinomatous neighboring lymph nodes were removed. Rupprecht also believes that carcinoma of the vulva, as a general rule, is one of the less malignant types. But in a 50 year old woman with a carcinoma of the nympho-labial fold there was no ulceration, but regional molluscum-like nodules and infiltrated inguinal lymph nodes; at autopsy, four months after the patient first visited her physician, the author saw many metastases in the lungs, the pleura, the myocardium, the thymus,

the kidneys, and a liver weighing 2400 gms., filled with cystic nodules (cornifying squamous epithelial carcinoma). Occasionally there is a carcinomatous ulcer on the side opposite to the primary growth; these, as well as the masses which grow spontaneously in the vagina and vulva in uterine carcinoma, have been believed to be "vaccination" metastases (see pp. 1697 and 1680); they are usually at first subepithelial, and discontinuous (metastatically) from the blood or lymph channels, or they are not true metastases at all, but merely a continuation of the original growth (see also Frankl, lit.). Otherwise metastatic tumors are not often met (lit. Grafenberg).

Cyst Formation in the Vulva.—Retention cysts of the sebaceous glands are called milium or comedo of the vulva; larger ones atheromas. Dermoid cysts are seldom seen. Old hematomas become (false) cysts and are often found in the labia majora. Cysts may arise from dilated obstructed parts of the canal of Nuck, and are called hydroceles of the canal of Nuck, (see Foderl for hydrocele muliebris). Cystic adenofibromas or adenofibromyomas of the round ligament may arise from fetal kidney rests (see p. 1661). Cystic lymphangiomas occur in the labia majora. For cysts of Bartholin's glands, see below. Pick (lit.) has described a pedunculated adenocystoma papilliferum polyposum, which he believes arose from entodermal epithelial inclusions from the sinus urogenitalis, (see also Ehrlich). For the older literature, see Orth.

Changes in Bartholin's Glands.—These mucous glands are as large as beans and empty into the vestibule through openings the size of a pin point on both sides of the hymen. They are the analogues of the bulbourethral glands (Cowper's glands) of the male (p. 1457).

The most important change occurring in them is the so-called Bartholinitis. This may be of a simple catarrhal or purulent nature, and in many cases results from gonorrhea. The process may be confined to one of the ducts, or it may involve the gland itself; pus then accumulates in the gland or the entire gland softens into an abscess.

This is manifested by a swelling at the base of the labia majora (the surface where the two lips are grown together) and a bulging in the median line. The parts become edematous and red. The process may remain stationary for years, or the pus may find its way to the surface, occasionally toward the rectum, or even into this structure (see p. 845). Gonorrheal Bartholinitis may be primary, or, more commonly, secondary to a vulvovaginitis, endometritis, urethritis. Colombini found gonorrhea in two thirds of the cases.

Suppuration of the glands when the duct is obstructed leads to the formation of a cyst containing mucoid watery fluid and perhaps of excessive size. Simple retention cysts of the ducts and acini also occur but are

comparatively small and due to inflammatory obstructions (v. Recklinghausen), as from scars after injuries at childbirth (Kustner), etc. The author reported a case (a 27 year old woman) with bilateral cysts as large as hen's eggs and lined by cylindrical epithelium; the excretory ducts were egg-shaped, transparent, and contained water-clear contents; the gland body itself was situated on one part of the wall like a small cap. Chronic productive inflammation leads to almost complete disappearance of the gland. Adenomas are rare (Coen). Squamous cell carcinomas or, more rarely, cylindrical cell carcinomas, have arisen from these glands. They appear as nodules, occasionally ulcerated, and progress to deep craters (see Mackenroth, Chaboux, Graham); Pape observed a case in a 91 year old woman; Tobler (lit.) saw a colloid cylindrical cell carcinoma. Sitzenfrey observed a cornifying squamous cell carcinoma of the excretory ducts in a 29 year old woman.

The Pregnant and the Puerperal Uterus and the Ovum

The Changes Undergone by the Uterus during Pregnancy. Decidua. Immediately following the entrance of the impregnated ovum into the uterus, there begins an increase in the number and size of the fibers until they are about four times their breadth and sometimes their length. The blood vascular apparatus keeps pace, and the mucosa produces the decidual membranes (also called caduca, perishable skin) which enter into intimate relationship with the chorion.

The upper one third of the cervix, *i.e.*, the part from the internal os downward to where the characteristic cervical mucosa begins, and upward to the lower uterine segment, takes part in this decidual change, since it is covered by typical uterine mucosa; the purely cervical part (the lower two thirds), containing characteristic cervical glands, participates only occasionally (Hohmeyer, Aschoff; see also Blumberg and lit. by Nurnberger). To contrast it from the body of the cervix, the upper one third was called the border zone or the narrow place (J. Veit), later, the isthmus, or the isthmus segment (Aschoff, see also Pankow, E. Moritz, H. Sternberg). Others believe this division into three parts, *i.e.*, the body, cervix and parts between the two, is unnecessary (for example Skutsch, Zweifel), and they retain the older division into the uterine body, the lower uterine segment and the cervix. See also Barbour.

The deciduae are differentiated into three parts: (*a*) the parietal decidua or the decidua vera; this is the largest and clothes the free inner surface of the uterine cavity; (*b*) the decidua capsularis or reflexa; this covers the ovum, consequently lies between the ovum and the uterine cavity; according to the older views it arises because the decidua vera at the border of the site of implantation grows over the surface of the ovum,

but according to newer opinions it closes off the intervillous blood spaces from the uterine cavity, (see p. 1711 under implantation of the ovum); (*c*) the decidua basalis (serotina) is at the situation on the endometrium where the ovum is implanted (is therefore peripheral to the egg); its villi are attached earliest and most firmly to the maternal tissues, inasmuch as they destructively enter as far as the spongiosa, while at the same time they cover the basal decidua with a thick layer of fetal cells (the so-called fetal decidua cells). From about the fourth month onward the capsularis is tightly grown together with the vera; in this way the lumen of the uterine body disappears. The capsularis then disappears completely (through stretching and necrosis), and the chorion laeve, also called the chorion (see p. 1711), comes to lie in immediate contact with the vera.

Histology of the Deciduae.—The changes which the mucosa undergoes to become characteristically rough and nodular, involve all its parts. In the fourth month, the deciduae reach their greatest thickness, that of about 1 cm. The glands elongate and dilate. The epithelial cells of the surface and of the glands (with the exception of those at the fundus of the glands) become lower; there are all sorts of transitions from cuboidal to endothelial-like cells, so that here and there the glands may even be mistaken for lymph vessels. The cylindrical ciliated epithelium disappears. The rounded and spindle cells of the interstitial tissue become large epithelial-like, spindle or polygonal cells rich in protoplasm, the so-called decidual cell. The cells of both the glands and the stroma during the first few months of pregnancy contain a large amount of glycogen (Marchand), which is particularly interesting, since the formation of the deciduae is in all probability a more emphasized, *i.e.*, a direct prolongation, so to speak, of the premenstrual condition of the mucosa (see p. 1619). In menstrual changes there is much glycogen (see Wegelin). According to Todyo, the decidua stores up the glycogen brought to it by the ovum. Uleskow-Stroganoff believe that the large cell decidual tissue is in part made up of epitheloid cells which come from fixed connective tissue cells by hypertrophy and hyperplasia. But another source is from an increase of the mononuclear cell (lymphocyte), which is already found in the mucosa or wanders into it. This cell is said to act protectively towards the organism by means of its phagocytic powers. The decidua vera (parietal) may be divided into three layers (Langhans). (*a*) The inner layer (compact cellular layer), closest to the ovum, shows the most marked growth of the interstitial tissue, which, with the exception of the blood vessels, originates almost entirely from decidual cells; it contains the openings and the lower portions of the glands which run a straight course directed almost perpendicularly to the surface; these often have a narrow lumen; the openings are pushed further from each other and pulled out in

a funnel-shaped form. (*b*) The middle layer, cavernous, ampullar, alveolar or spongiosa, lying nearer the muscle, contains glands which develop a broad, wide, flat network almost parallel to the surface; the direction in which this network lies, however, is not due to pressure exerted by the ovum, for it is found in the deciduae in the empty uterus in ectopic pregnancy. These glands are lined by low to flat epithelium, the individual cells swollen, full of mucus, or undergoing fatty degeneration. The interstitial tissue is sparse; here and there, thin septa develop between the glandular spaces. (*c*) The innermost basal layer bordering on the musculature contains the fundi of the glands in a compact ground substance; this epithelium is not flattened. This layer takes very little part, perhaps none at all, in the actual decidual changes. In this condition, as well as in the resting mucosa, the use of special stains (silver impregnation, according to Bielschowsky) demonstrates that there is a fine intercellular reticulum (Hormann). Toward the end of pregnancy, the deciduae gradually undergo a necrobiotic process, mainly in the form of a coagulation necrosis. The cell borders in the later stages of pregnancy become obscure; the nuclei do not stain. Fatty degeneration plays an inconstant and unimportant role (G. Klein). During childbirth the layers of the deciduae become separated, usually in the more or less well developed cavernous layer, occasionally higher or deeper (see Baisch). This is supposedly brought about by the entrance of blood into the vessels of the spongiosa immediately after the birth of the child, thus tearing the thin septa and loosening the placenta (Frankl, see also Hiess). After this separation, raw remnants of deciduae hang from the fetal membranes (chorion laeve and the inner surface of the flat amnion), *i.e.*, from the outside of the chorion; they consist mostly of hyalin degenerated parts of the compacta and also of larger tags of spongiosa. The development of a new mucous membrane during the puerperium takes place from the remaining blind ends of the glands and that part of the interstitial tissue left near the loose layers of the muscularis. In the capsularis, which is built somewhat like the basalis (serotina), and in general similar to the parietalis (vera), the glands run more obliquely or parallel to the surface. Toward the fifth month the capsularis is so stretched by the growth of the fetus that both glands and blood vessels are lost; nutrition is then obtained by way of the parietalis with which the disappearing thin membranous capsularis is combined.

The decidual membranes form the maternal parts of the fetal membranes.

Decidual nodules, *i.e.*, decidua-like overgrowths are often found on the peritoneum directly under the covering cells (see p. 870), in the omentum, the appendix, in the ovaries, the tubes, the pelvic lymph nodes, and par-

ticularly in the hypogastric glands (Geipel, lit., Haendly). It is thought that they are stimulated by the activity of irritant substances which arise in the pregnant uterus or perhaps in the ovary. R. Meyer believes, however, that inflammation plays a much greater part in the causation of these structures. See Hofbauer for histological peculiarities in other organs such as the vagina, bladder, even the laryngeal mucosa (?). For the experimental development of ectopic deciduae in the serosa of the uterus, see Unterberger.

For the causes of deciduae formation, see the theories and experiments of Leo Loeb, who believes that it is due to the influence of a chemical substance coming from the corpus luteum.

B. **The Membranes of the Fetus, Amnoin and Chcrion. Placenta.**— See O. Grosser for the comparative anatomy and embryology and literature.

The embryo lies in a cavity filled with the liquor amnii; its walls are made of two easily separable membranes; the inner is called the amnion, the outer is called the chorion.

The amnion is an extraordinarily tender, transparent, connective tissue covering, composed of a simple layer of flat epithelium, (the secretory functions are discussed by Mandl); from the inner surface of the placenta it passes out as a covering over the umbilical cord. At the navel of the fetus the epithelial layer blends into the epidermis, the connective tissue layer into the cutis of the fetus.

Outside of the amnion is the chorion (villous coat), which is also transparent and arises from a mesodermal connective tissue layer bringing in blood vessels (endochorion); it is covered externally with many layers of epithelium from the trophoblast. The trophoblast has at first a flat surface but then develops a rugous appearance, and finally there arise buds free of mesoderm, the so-called primary villi. The endochorion with the allantois passes through the pedicle of the allantois to the outer coverings of the ovum. In the beginning, the chorion is covered everywhere by villi (chorion frondosum); each villus is covered at first by several layers and later by only one layer of simple epithelium; in its connective tissue stalk it contains an end branch of the umbilical artery which then anastomoses with a small vein. See p. 1710 for the further appearance of the chorion at birth. We must now go backward into the embryological development and study more closely the lodgment of the ovum. Accurate details of this process are known as they occur in the guinea pig (Graf Spee, Biedl, Peters and Hofstatter), but for the human there are as yet few comprehensive studies; among them may be compared the observations of H. Peters, Jung (lit.), Bryce and Teacher, Strahl and Beneke, Todyo, Boerma, Linzenmeier, Keibel, Schlagenhaufer and Verocay, Temesvary, lit. According to these authors, the ovum sinks

into the superficial layers of the mucosa. The uterine epithelium is destroyed where it comes in contact with the ovum. The opening marking the spot at which the sunken egg passed into the mucous membrane is then closed (connective tissue or plug of blood, Peters) to the uterine cavity; this thin covering is called the decidua capsularis or the so-called reflexa. It is stretched more and more as the ovum becomes larger and the villi disappear entirely; concerning their disappearance with the chorion laeve, see p. 1708. For illustrations see, among others, O. Grosser. According to a newer view there is not one-sided erosive activity of the fetal parts, but a cooperative activity of both fetal and maternal tissues in the development of the placenta, (d'Erchia).

The Epithelium of the Villi.—As investigations on very early stages of the placenta of animals and in very young human embryos have shown, (Langhans, Strahl and others), there is an epithelial covering consisting of two layers; the lower layer contains clear, cuboidal cells (Langhans' layer), and is undoubtedly of fetal, or more conservatively, of trophoblastic origin; the outer protoplasmic layer is rich in darkly staining nuclei, contains fat granules and a striated border; the latter is called the syncytium (Bonnet). There are two opinions concerning its origin; the one is that it comes from the maternal part, in other words, that it is changed uterine epithelium (Langhans, Strahl, Kossmann, Merttens and others), and the other that it is of fetal origin (Kolliker, Leopold, Ruge, Minot, His, Peters, Kollmann, Maximow, Graf Spee, Leopold, Bonnet, Jung and others). The latter seems to have the more followers at present, and therefore it is thought that both layers arise from "primary ectoderm" (trophoblast) of the germinal vesicle. Strahl and Beneke, however, believe that this question cannot be answered with the material we have at present. In the very earliest stages the villi are not covered by cell layers of syncytium, but by the trophoblast, which is then called fetal ectoblast (Peters, Siegenbeck and Heukelom, as well as Hitschmann, Lindenthal and Franz); then there follows a differentiation of the trophoblast into two layers. It is, however, questionable if the trophoblast is brought into such close relationship with the ectoblast (see Mewes, Schlagenhaufer, de Snoo), since the trophoblast is the earliest outer shell of the germinal vesicle; from the embryonal cells of the latter, however, there first develop the three germinal layers. The fetal mesoblast comes into relation with the trophoblast, and forms the stroma, while the trophoblast provides the coating of the chorionic villi, then in its turn separates into Langhans' cells and syncytium. According to Broman, the trophoblast in a wider sense, should therefore be considered part of the ectoblast. It may be stated with confidence that the uterine glands are not the source of the covering layers (Kollmann and Maximow). In later stages (the

second half of pregnancy), the epithelium of the Langhans layer becomes flatter and no longer is present as a continuous membrane on the villi; in the ripe placenta it can be found usually only in traces (large oval nuclei between the covering layers and the stroma of the villi; Merttens). As a rule, the Langhans' cells disappear from the fourth month onward and with them the proteolytic enzymes of the cells, which makes the chorionic villi lose their ability to digest protein, (Grafenberg). There is then seen only a simple, irregularly thick, strongly staining layer of epithelial cells with their borders obscured (syncytium), but their nuclei rich in chromatin.

At the end of the second month all of the villi disappear (chorion laeve), with the exception of those which lie inside of the basal deciduae; these undergo vigorous growth and together with a chorion plate compose the fetal placenta (chorion frondosum). The complete placenta, the organ of respiration and nutrition of the fetus (for more details see Hofbauer), which may also be the seat of stimulation for parturition pains (Guggisberg, Ludwig), is composed of both a maternal and a fetal part; the former arises from basal plates which come from the deeper layers of the decidua basalis and the so-called decidual pillars which are responsible for the rough septa in the placenta. (Concerning the internal secretion of the placenta, see Halban, Schickele, Acconci, B. Wolff, lit.)

The relation of the chorionic villi with the maternal tissues, which makes possible the development of a circulation for gas and metabolic exchange between the fetus and the mother, was formerly believed to be as follows (Grosser, Frank, lit.): at the situation where the villi touch the decidua basalis, there occurs a vigorous growth of cell layers; buds appear which later take on a mesodermal stroma. The cells wander into the mucosa (see p. 1716) and spread out on the surface of the decidua basalis as the so-called suction plate (see p. 1709). The uterine epithelium which shortly undergoes the syncytial changes (they occur in extrauterine pregnancy also, see p. 1604) is destroyed by the advance of the cells and of the villi covered by the cells. As the latter force themselves into the mucosa, they erode the blood vessels of the serotina, and blood is poured off into the intervillous space, which is at first between the vesicle and the surface of the serotina, *i.e.*, between the fetal and the maternal tissues, and through it the villi force themselves from the ovum to the maternal tissues. As the intervillous space is marked out, there follows a certain amount of separation between the fetal and the maternal tissues, so that the boundary line is changed into the so-called Nitabuch's fibrin stripes (according to Grosser it is more accurate to call them fibrinoid stripes) in which both maternal and fetal boundary cells are destroyed. The importance of Nitabuch's stripes is said to be resident in an antiferment which prevents

too vigorous and aggressive activities of the fetal villi. In placenta increta, but also in the normal physiological invasion into the uterine wall (see p. 1716), this is broken through. These are also spoken of as hyalin-necrotic stripes, and it is assumed that they are the expression of a softening of the tissues of the decidua through the activity of digestive ferments from the syncytium, and that later this tissue undergoes liquefaction. Later, there appear more and more branched villi which dip into the intervillous spaces, while on the other hand, still other villi, which arise as broad stems in large number from the chorion, insert into the serotina as the so-called trunk or attachment villi. Inasmuch as arterial blood flows into the space through small openings while venous blood flows out through other openings, there arises the so-called intervillous circulation; the maternal blood flows therefore through extravascular channels and plays over the fetal villi. The arteries and veins are connected only by a system of anastomosing spaces, which are entirely surrounded by the fetal villi (Kollmann).

On the other hand, according to Peters, Siegenbeek van Heukelom and others, the intervillous circulation is thus constructed: the chorionic layer of ectoblast (trophoblast) which lies directly in contact with the maternal tissues opens the maternal blood capillaries by erosive action. The onrushing blood then completes the spaces, the lacunae, in the tropho- or fetal ectoblastic layers; these spaces are therefore entirely surrounded by fetal tissue. According to Jung, they are dilated by and during the first growth of the villi (lit. by Hinselmann). Partitions of cells remain between the lacunae and into these there grows secondarily from the germinal vesicle, (Peters, Jung), the mesoblast with its vessels. These constitute the primary villi; later, there develop branches which also dip into the blood spaces. According to Duval, however, the maternal vessels grow into the ectoblast of the fetal villi, the vessels dilate to blood spaces, and as a result, the chorionic villi gradually hang free in the maternal blood, *i.e.*, within maternal vessels. If this viewpoint is adopted, it seems necessary to believe that the syncytium is composed of maternal endothelium (Pfannenstiel), and while a number of authors do believe that this membrane is "endothelial" and, as stated, is divided into two layers covering the chorionic villi (Merttens, Keibel, Langhans), most authors, such as Paladino, Kollmann, Maximow and Jung, believe otherwise.

The (fetal) smooth inner surface of the placenta is covered by the amnion. After the placenta is born the serotina covers the outer surface with a raw layer about 2 mm. thick, the basal layer (Winkler), from which arise wedge shaped septa (decidual pillars) which divide the placenta into lobules (cotyledons), and which are intimately connected with the chorion

to form the subchorionic occluding plate. At the edge of the placenta, where the basal layer and this closing plate join, there is a prominence. In the middle layers of the placenta the decidual tissue is forced apart into little islands by the intervillous blood spaces. These may be injected from the aorta or from the uterine artery (Waldeyer, Bumm), and are therefore shunted about between the utero-placental arteries and veins. The utero-placental veins which empty the intervillous blood spaces, develop into a network which runs almost parallel to the decidual surface of the placenta, and which empties in part toward the edges of the placenta into a ring-shaped venous sinus (border-vein), and then sinks into the thin-walled venous sinus of the muscular part of the uterus.

The normal loosening of the placenta and the membranes takes place in the spongy layer of the deciduae (see p. 1710). A part of the latter is expelled with the placenta and covers the villi and intervillous spaces as a thin layer; the other part remains behind with the torn utero-placental vessels. The fundi of the glands are left in the uterus even at the place of attachment of the placenta (Leopold, Hiess).

The name "placenta accreta" (grown tight) or "increta," describes the condition when the placenta is attached abnormally tightly, either partially or in toto. It has never been observed in primipara but often habitually in multipara. The chief cause is an insufficient development of the deciduae as a result of destruction of the mucosa by many pregnancies (Zweifel), or injuries to the endometrium by abrasions, vaporization, repeated manual delivery of the placenta, etc., (Schweitzer, lit.). The chorionic villi after tearing apart the poorly developed deciduae, force themselves more or less deeply into the musculature, even to the serosa (Wegelin, R. Meyer, M. B. Schmidt). According to Frankl, a cause is lack of room, which does not allow of sufficient spread; and this is compensated for by deeper growth. The importance of an abnormal insertion is emphasized by Baisch. If the uterine musculature is used up by repeated placentation, so that the villi reach under the serosa, the uterus may rupture during childbirth (Schwendener, Sachs); the author observed such a case of spontaneous perforation in a 40 year old woman who had given birth to five children; during childbirth fatal hemorrhage into the abdominal cavity took place through a tear about as big as a finger through which the villi protruded; about $2\frac{1}{2}$ liters of partly fluid and partly coagulated blood were found in the abdomen (case reports, lit. and details by Dietrich). An adherent placenta caused by disturbance in the muscular expulsion apparatus, *e.g.*, placenta in the opening of the tubes, must be differentiated from placenta accreta.

The theories concerning placenta praevia are varied. Aschoff says there may be a placenta praevia simplex and a placenta praevia isthmica,

according to whether the placenta in part or as a whole is situated in the isthmus (see p. 1708). But according to Zweifel, it is always inserted in the body, (but see also Bayer, Jolly, Nurnberger, Ruge I, Schweitzer); it may, however, be in the lower portion and separate a muscular lamella of the cervical wall, thus becoming a placenta increta and protruding into the lumen of the cervix (placenta praevia cervicalis, see Krause, lit.).

Placenta Exo- or Extra-chorialis.— If the placenta is too small, the villi spread in the uterine mucosa beyond the limits of the placenta covered by the chorionic membrane, and thus there is an exo-chorionic zone (according to R. Meyer). The placenta marginata also, in which the chorion laeve with the decidua capsularis does not, as normally, leave from the outer border of the placenta but 2 to 3 cm. inward from this situation, is, according to R. Meyer, a placenta extra-chorialis; in this condition the vessels running on the surface of the placenta are sharply kinked at the internal ridge of the margin. See Hinselmann concerning the changes in the decidua parietalis in exo-chorion pregnancy caused by pressure, pushes or other trauma on the part of the fetus.

Particularly in the first half of pregnancy (Schickele), many fetal cells wander and break through the Nitabuch stripes into the maternal mucosa (deciduae) and musculature; these chorionic cells find their way particularly into the venous system, by burrowing under the endothelium and breaking into the lumen. These have been called chorionic wandering cells (Marchand), but others believe most of them, on the contrary, express a decidual reaction of the maternal tissues (Pinto, Fellner and others); R. Meyer (lit.) believes in the chorionic nature of these polymorphous cells but not on their ability to wander; he thinks they are present only as a result of continuous cell growth. According to Pels Leusden, this invasion of the chorionic cells reaches its height between the third and sixth months. The degree of infiltration is quite variable. (For literature concerning chorionic cell invasion, see Nurnberger, also Schiller.) The shape of the cells is much changed; many become multinucleated giant cells with vague boundaries. Not only single cells but also cell masses, many with syncytial giant cell-like ducts, even villi, may enter the veins in normal pregnancy and, particularly in labor of long duration are they loosened and carried onward through the blood stream (Schmorl). According to Poten, this deportation (Veit) of chorionic villi probably occurs in every pregnancy (see also Hitschmann). Normal deported villi and single cells are destroyed without giving rise to symptoms. This is probably due to the solvent action of cytolysins in the sense of Ehrlich.

According to Ehrlich's theory, the organism has the ability to develop antibodies, the so-called cytolysins, against strange cells and cell products.

Veit has called these antibodies against deported cells and cell products in the blood of pregnant women, syncytiolysins (see Wormser, lit.). It is believed that when these antibodies are lacking, the circumstances favor a rapid overgrowth, *i.e.*, formation of a malignant tumor (syncytioma or malignant chorion epithelioma). (See Schmauch, who optimistically believes that successful therapy of this tumor may be practiced by immunization against its specific cells.) After childbirth these giant cells usually disappear from the uterine musculature in eight days, but they may persist for weeks, until they finally degenerate and become hyalinized. If, after childbirth, they continue to grow, and grow vigorously, it is an indication for extirpation of the uterus (see under malignant chorion epithelioma, p. 1739). Pels Leusden believes these giant cell syncytial epithelial masses are important in the regeneration of the uterine epithelium, but others cannot share this belief (Aschoff). Their importance is still undetermined. These so-called placenta giant cells, which are histogenetically of the same origin, may also be found in the lungs (capillaries and smaller arteries) in puerperal eclampsia (Schmorl, Lubarsch and others). It is not likely, however, that they have any particular significance in this condition (Pels Leusden). The tetanic convulsions of the body probably aid in loosening them. Sometimes decidual giant cells are also observed (see W. H. Schultze, Oberdorfer). See p. 388, concerning giant cell embolism in the bone marrow.

The Puerperal Uterus.—Immediately after childbirth, the internal surface of the uterus is of a blue-red or yellow color corresponding to its lining of deciduae. It is ragged and shows depressions representative of the spaces in the decidua ampullaris (see p. 1710), in which the separation of the placenta usually occurs. There is no covering of surface epithelium, and the decidua with the exception of the places containing the fundi of glands, is a true wound surface. This excretes lymph and blood, the lochia, and is cleansed by expulsion of the ragged masses of deciduae which undergo necrosis or fatty degeneration and are discharged with the lochia. There is also emigration of many leucocytes which mix with the tags of tissue and the lochial fluid and no doubt through their ferment take part in the solution of the perforated tissues. At first the lochia are bloody and profuse; they gradually diminish in amount, become dirty brown-red, later yellow-white, and in ten days or more fluid, like serum. The discharges usually cease after about six weeks (Reifferscheid). Lochia from the uterus are, as a rule, free of mucus; those coming from the vagina are more apt to contain this substance (for more details, see p. 1751). Healing, which results in complete replacement of the mucosa, takes place from the spindle cells of the interstitial tissue and blood vessels, and from the fundi of the glands which grow from the deeper layers of the

mucosa. In about eight days, these epithelial cells have made an entirely new surface epithelium as well as new glands; the process is completed in about three weeks (Wormser, lit.).

The place of attachment of the placenta juts into the cavity of the uterus like a plateau; on it can be seen the venous sinus (of the serotina and the neighboring musculature), partly thrombosed even before the birth, and the remnants of the thin walled utero-placental vessels; the serotinal tissue is bloody. For the most part, however, this takes place post partum. The thrombi are organized, the vessels destroyed, the protrusions disappear; after six weeks there can still be seen a small amount of protrusion and brown pigmentation of the inner surface; in three months the situation of the placenta can no longer be found. According to Frankl and Stolper (lit.), the chief role in the obstruction or closure of blood vessels after birth is played by a connective tissue cushion—prepared during pregnancy by decidual changes in the vessel walls—which closes the vessels like valves; such valve formation has been seen in the vessels of the umbilical cord also (see p. 116; see also Heckner, lit.). This naturally assists in the thrombotic organization processes. See Goodall and also Buttner concerning the involution and regeneration of the post partum uterus, particularly of its blood vessels. See Labhardt, lit., concerning the anatomical explanation of post partum hemorrhages.

The cervical canal quickly returns to normal; injuries incurred heal very quickly. After three days, only a single finger may be inserted. After ten to twelve days, the external orifice contracts to its normal size. The external os becomes an oblique slit and the cervix is no longer conical.

The involution of the musculature of the puerperal uterus was discussed on p. 1642. The weight is reduced from about 1 kilogram to about 100 grams. It may be mentioned, that, according to Sanger, the muscle fibers during involution become more and more the seat of fatty degeneration (see Adachi and also Huguenin), but do not disappear in toto; they become smaller by absorption of the fatty masses, and atrophy takes place, but the nuclei are preserved for the future, so that a new regeneration of the muscularis may occur. The glycogen collected during pregnancy in the muscle fibers and vessels quickly disappears during the puerperium (Broers, Gierke). According to O. Horn, the elastica, which gradually disappears during pregnancy, again shows significant increase. It is worthy of notice that in focal situations in the uterus there appear cross striated muscle fibres during the puerperium (metaplasia?); see Girode, Nehrkorn and Hofbauer.

Diseases of the Decidual Membranes.— If a chronic hyperplastic or gonorrheal or, as is sometimes assumed, a syphilitic endometritis, is

present before the occurrence of pregnancy, or if it develop during its course, there arises a diffuse or nodular polypoid thickening of the deciduae (diffuse hyperplastic —or polypoid—or tuberous decidual endometritis).

The latter often occurs in lues (Virchow). From overgrown uterine glands, there may develop pedunculated vesicles which are prominent in the separated decidua vera. The thickened decidua is richer in blood vessels and is infiltrated by small cell masses, fibrous bands and fibrinous stripes. These changes are usually recognized only in the prematurely born fetus. The products of abortion may be expelled with the entire deciduae as a three cornered sac, or the whole hyperplastic decidua remain in the uterus; the placenta is expelled, and later there follows the expulsion of a three cornered, thick walled, empty decidual sac. But pregnancy may proceed to normal term, in which, among other things, a hyperemesis gravidarum may arise; in the placenta, however, are often found the so-called infarcts (p. 1728), and the fetus may show more or less malformation.

A true inflammatory change in the endometrium inhibits either conception itself, or the proper embedding of the ovum, because it prevents the internal adherence of the vera and reflexa, whereby the serotinal anlage is small and pedicle-like. Death of the fetus, abortion, or the formation of a hydatidiform mole may follow.

In hydrorrhoea uteri gravidi, which is quite uncommon and usually begins in the third to the fourth month, the adhesions between the decidua vera and the reflex are not formed, and the catarrhal secretions from the diseased vera coming from the endometrium may flow out of the uterus intermittently.

See Hinselmann concerning changes in the decidua parietalis (vera) in exochorial pregnancy.

See p. 1632 concerning decidual endometritis remaining after abortion.

Disease of the Fetal Membranes. Diseases of the Amnion.—The amnion is the innermost membrane surrounding the fetus (in truth a part of the wall of the fetus, a prolongation of the skin of the child, see Strassmann); it consists of an epithelial (ectoderm or epiderm) layer, which, according to Forssell, is made up of two rows of low basal cells and higher cylindrical cells with rod-like edges, and a tender connective tissue, the latter the outer mesodermic layer of the body. The epithelium of the amnion probably takes part in the secretion of the amniotic fluid (Polano, Mandl, Bondi, Forssell), although some authors believe the fetal urine (see Gusserow, Saltykow, lit.) makes up the greatest part, a view, which G. A. Wagner does not believe possible under physiological conditions (see also B. Wolff, lit.). According to Forssell, at least after its formation,

the placenta produces most of the amniotic fluid. This fluid is a source of protection against injury to the fetus and provides it with a medium in which it can move freely.

When the amount of amniotic fluid reaches 1 to 1½ liters, the condition is called **hydramnon;** there have been reported cases with 20 liters (Schneider).

The placenta in this condition is often heavy, large, even three times the size of the normal organ, and quite edematous. The increase in the amount of amniotic fluid may come from the fetus or mother. In one case hydramnion is found in circulatory disturbances of the mother which are accompanied by edema and in which there is an increased transudation from the deciduae into the amniotic cavity. On the part of the fetus there may be circulatory disturbances in the umbilical vein following nodular growths, torsions, or stenoses, and these lead to vigorous transudation from the fetal circulation, and to heart failure, stenosis of the duct of Botalli, as well as to hepatic and pulmonary thickenings such as occur in syphilis. Perhaps in the etiology of hydramnion we may also consider increased urinary excretion on the part of the fetus (see under the circulatory conditions in hydramniotic one ovum twins), inflammatory changes in the amnion, or, perhaps, diminished resorption of the fluid. In conjunction with hydramnion there are often malformations of the fetus such as hydrocephalus, anencephalus, spina bifida, pes varus, extrophy of the bladder, hare-lip and cleft palate as well as hydrops of the entire fetus. In a 47 cm. long, cyanotic, edematous fetus, the author saw changes similar to those described by Schridde, viz., colossal hematopoietic masses and hemosiderosis of the liver, prominence of the myeloid tissue in the thirty gram spleen, hematopoietic masses in the kidneys; the placenta weighed 1500 gms.; syphilis as a cause was excluded. Sitzenfrey saw an analogous condition in twins with congenital acute nephritis and universal edema. An acute form of very high grade hydramnion occurs in rare cases in one ovum twins (they always have a single chorion), in which the uterus even at the fourth to the fifth month may have the appearance of one at full term. The sac of only one of the twins is hydramniotic while that of the other one contains the ordinary amount of fluid. The former fetus is better developed; its heart and usually its kidneys are hypertrophied; there is stasis in the inferior vena cava; ascites and anasarca may also be present. This hydramnion may possibly be explained thus: the vessels in the placenta of one chorion twins form anastomses between the umbilical cords, but if they do not function properly, or if the hydramniotic twin, on the contrary, receives too much blood from the other twin, there results an overfilling of the circulation of the former which leads to stasis in the umbilical vein, hypertrophy of the heart, and an increased amount

of urine with a high percentage of urea in the hydramniotic fluid (Kustner); (see also Kaltenbach). See Seitz for the literature concerning hydramnion.

An abnormally small amount of amniotic fluid (oligohydramnion) in the first few months of fetal life may hinder the development of the embryo in a number of ways, particularly by decreasing the size of the cavity, so that insufficient loosening of the amnion brings about the production of adhesions between it and the surface of the body (Simonart). Later, when the amniotic fluid increases in amount these adhesions may be pulled out into bands, or threads, (see fig. 926), although it is believed by some that these amniotic strands are due to inhibitory disturbances in growth. They may lead to adhesions, especially between the fingers and the toes—a form of syndactylia—or between the limbs, from which scars may result, or self amputation with terminal scar formation; or, they may give rise to defects in the skin, or to the formation of clefts, *e.g.*, in the region of the face (lit. by Sitzenfrey); if the adhesions are broad, they ofttimes lead to ectopy of the abdominal organs, umbilical hernias, hernias of the brain, etc. (Kustner, Kummel, Klaussner, Haymann, lit., Theodorow, Schwalbe, Scheffen, Stephan, Murk Jansen, lit.). Thus amniotic bands or the pressure exerted by them may be the most important causes of exogenous malformations.

According to Graf Spee whose investigations were made on rodents, (direct observations concerning the first development of the amniotic cavity in the human have not as yet been made), the amniotic cavity is first laid down as a solid mass of cells. In the fourth week the size of the embryo increases until it occupies almost the entire cavity (Minot). Then the cavity is stretched to a considerable space by the collection of fluid, but in places here and there it is still in contact with the fetus. It may be that in the earlier stages, as the sac fills, these places do not separate, and thus there appear bands, (Bardeleben); or it might be possible that these bands arise from adhesions following an amnionitis occurring about the end of the fourth week (see Strassmann). This latter assumption approximates Simonart's conception of their origin, *i.e.*, as the result of secondary growths. The inflammatory origin, however, is disputed by Marchand, Schwalbe, Wolff and others. The intraamniotic pseudomembranes (Marchand, lit.) are fibrillar connective tissue membranes independent of the amnion; they almost cover the fetus, and originate, perhaps, from organization of small hemorrhages coming from and into the chorion. According to Marchand, these membranes are responsible for the amniotic growths or adhesions. If the entire embryo is covered completely by pseudomembranous adhesions, serious malformations usually follow.

If the amount of amniotic fluid remains constantly at a very low level the entire development of the fetus is hindered. There follow extreme malformations such as monsters with legs united, cyclops, congenital club-feet, flat-feet and others (Kustner). (For the literature concerning the amniotic fluid, see Goenner.)

See Sitzenfrey concerning anomalies of the amnion, such as villous overgrowths of the epithelium, white plaque-like growths of the amniotic tissue, white wart-like vernix nodules, and umbilicated projections.

Diseases of the Chorion. Hydatidiform Mole.—In these structures, the chorionic villi are changed into transparent vesicles. The process

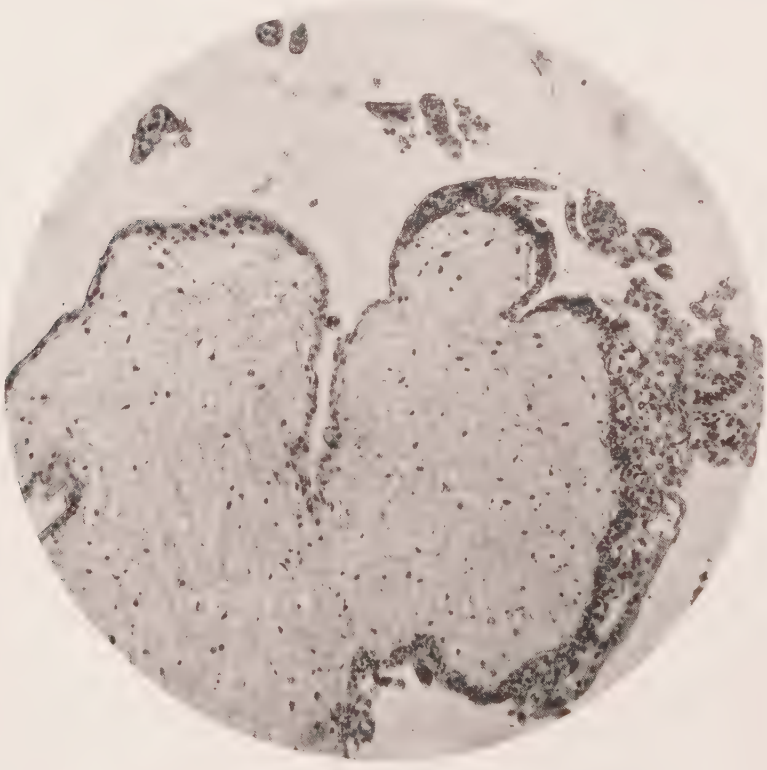


FIG. 865.—Hydatidiform Mole. × 130. L. H. #37.

may involve the entire chorion, since originally this contains villi over all its surface—and then no placenta can be recognized—or the process may involve the placenta alone or only isolated cotyledons; the latter is quite common in abortions and is important in considering chorion epitheliomas. Occasionally, isolated villi change to vesicular, rounded or spindle-shaped structures from the size of a grain of corn to a cherry, which are connected by thin threads of fibrous tissue so that the masses hanging together give the appearance of a bunch of grapes. The contour of the mole is pre-

served by a thick layer of deciduae, and when such a mole is born, it is held together by blood coagula also. It may reach a weight of five pounds or more.

If these changes take place in the first two months of pregnancy, the entire ovum is changed into a mole, and there can be found no amniotic

FIG. 866.—Villi of a hydatidiform mole with hydropic stroma, marked proliferation of covering cells and giant cell syncytial buds; woman 34 years old, bore 6 children; last menstruation 10 weeks before; 4 weeks before severe hemorrhages; curettage removed vesicular villi, blood clots, decidual tissue; woman remained permanently well. (Quite low power.)

sac, no fetus and no placenta (sterile mole). In a case of Sitzenfrey, the amniotic sac, because of hydropic degeneration, was mistaken for a large villus. If the changes in the villi occur later and are very extensive, the fetus dies and can be found in the amniotic cavity; the placenta can be identified partially or totally changed into grape-like masses. Usually the hydatidiform mole is expelled in the third to the fifth month, often

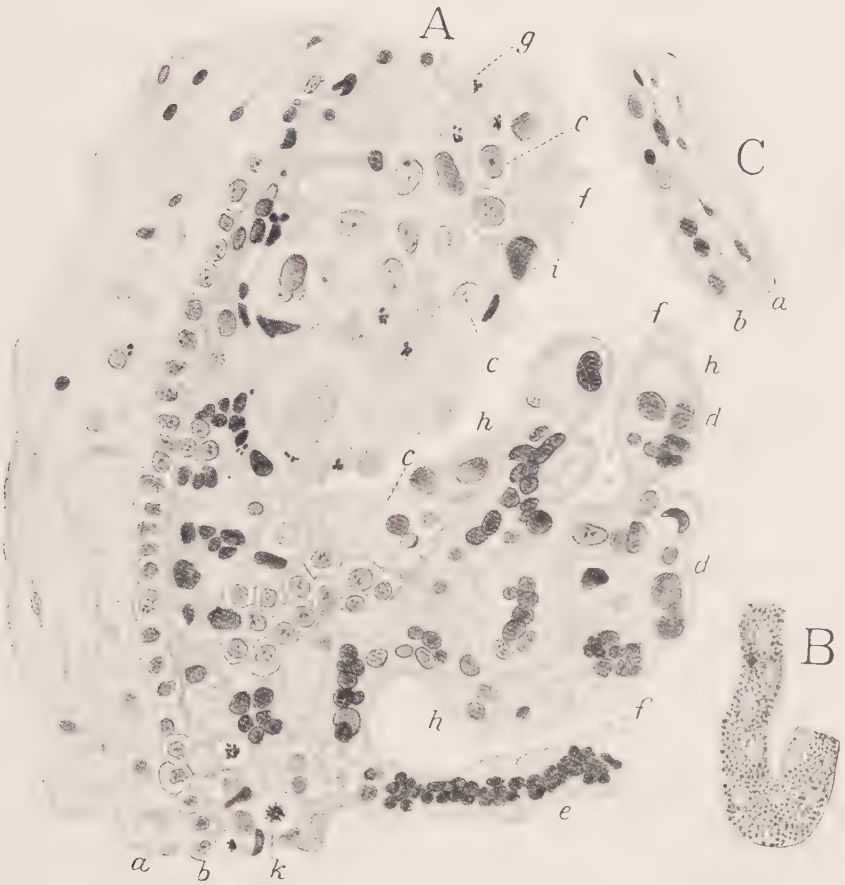
with severe hemorrhage. According to Seitz, a hydatidiform mole occurs about once in every five hundred child births. Hydatidiform moles of the tube are quite rare, (lit. by Risel). The etiology is uncertain and much in dispute (see Essen-Moller for complete literature). An abnormal condition of the endometrium, a "decidual endometritis," has been blamed, (see among others Stoffel, Seitz); on the other hand, there have been cases of twin pregnancy in which a hydatidiform mole has developed in one twin, and the other fetus was normal. This finding favors the conception of a primary disturbance in the ovum itself (lit. by Risel, see also Seitz). For the theories of Durante and others, see p. 1726. The disease as a whole is rare, but occurs relatively more often in older individuals; it is often accompanied by albuminuria, with nephritis and edema. The uterus grows with extraordinary rapidity. Then there occur hemorrhages and the expulsion of a thick mucus which probably comes from the pulling apart of isolated edematous villi. The opinion that lutein cysts of the ovary are a typical coincident finding (Koremer) is not verified by the experience of the author (see p. 1553). There also occur combinations with blood moles.

In the histological make-up, Virchow believed there was a mucoid swelling with an increase in the mucoid tissue, and hence called the process myxoma chorii. Langhans, however, opposed this view, and Orth pointed out that chemical staining with methylene blue or thionin does not show that the contents are mucus; the condition gives more of the impression of an edematous change. v. Franque, on the other hand, believes there is an increase of mucus, *i.e.*, a secretory anomaly of the Langhans cell layer.

Todyo found that the mucin reaction almost disappeared in the myxomatous large villi in well developed hydatidiform moles, while smaller villi, in the beginning of degeneration, show the reaction.

But, according to the investigation of F. Marchand and L. Fraenkel, the hydropic and the partially mucoid changes beginning in the center of the stroma and leading to bubble-like blowing up of the villi, are not the most important part of the disease; the changes begin rather in the epithelium of the villi, where there is a vigorous, more or less lawless, overgrowth with many mitoses, chiefly on the part of the Langhans cell layers (fetal; see figs. 866 and 867). The overgrowth may be very marked as illustrated in the case observed by Marchand, and in the case illustrated in figs. 866 and 867. Sometimes, the growth is much less marked, as Langhans has testified, and the author can verify. The size of the isolated villi, however, may be quite varied (fig. 866). The vigorous overgrowth is followed by degenerative changes in the cell masses, including the syncytium; the latter then shows its genetic similarity to

the cellular layer (see for example, fig. 868). These changes consist of hydropic vesicular swelling, and vacuolization with irregular placement of the nuclei. In many places the cell growth is transformed to a finely fibrous, almost anuclear, network (see fig. 866). Glycogen is present in the Langhans cells; in the syncytium there are many fat droplets (see



FIGS. 867–869.—Details of hydatidiform mole of fig. 866, from the edge of a villus; a, villus stroma with cells; b, Langhan's cells containing glycogen; c, these cells proliferated and enlarged, with vacuoles and very large nuclei in places; i, fatty degeneration and nucleus rich in chromatin; d, syncytium with vacuoles and large nuclei; e, syncytium with closely massed nuclei, rich in chromatin; f, red blood corpuscles, some blended into hyalin masses; g, leucocytes; h, striated border on syncytium; k, mitosis in a Langhan's cell and close by two other cells in mitosis. B. Piece of syncytium from another case (Flemming preparation); fat droplets and granules stained black brown by osmic acid; here and there visible nuclei; others covered by fat. C. Piece of covering of a villus of the same mole as A, which illustrates under b, the transformation of the layer of Langhan's into syncytium; nuclei enclosed still distributed at fairly regular intervals; striated border; below the syncytium at once the mesodermal stroma (a) of the villus with its spindle-cells. (A.B.C.— × 283.)

fig. 868). Degenerative changes predominate in the connective tissue of the avascular villi; the central parts undergo liquefaction, the peripheral

parts usually remain intact; some of the swollen cells undergo necrosis and their nuclei disappear.

According to Aichel, the hydatidiform mole arises because of the absorbing ability of the chorionic epithelium which persists, even though the embryo or the fetus be dead, or already absorbed. Communication with the fetus through the umbilical cord, is still intact though the fetus be dead. The retained material soaks through the villi. Durante believes there is a successive disappearance of the capillaries of the villi following an obliterative overgrowth of their endothelial cells (obliterating endocapillaritis; the cause, maternal toxins). Since the cellular covering of the villi remains intact, materials are absorbed from the maternal blood by osmosis, (provided, of course, that the maternal circulation does not stop), and the villi swell, because the absorbed material cannot reach the fetus when the vessels are obstructed. The overnourished syncytial covering grows not only to compensate the dilatation of the villi, but also widely beyond this stage. In a second work, Durante describes a mole as an "adenome villeux." The same explanation holds true for a sterile mole in which, of course, the fetal vessels are lacking in the villi. Another theory places the responsibility on a primary change of the ovulum, or its ectoderm (Marchand); another, on a primary alteration of the endometrium leading to a variance in the character of the villous epithelium (Veit). Hinselmann attributes the edema of the villi to a disturbance of the fetal vascular growth and describes proliferative changes in the inside of the villi of the hydatidiform mole.

Occasionally, large cells, believed to be wandering fetal epithelial cells (Neumann), are found in the stroma (Chaletzky), but Hinselmann and Marchand also attempted to show that these cells, particularly those found on the inner surface of the central cavities, arose as a result of the connective tissue overgrowth. It is particularly interesting that the growing swollen epithelial cell masses (particularly those of the Langhans cell type) by breaking through the fibrinoid stripes (see p. 1713) force their way not only into the decidua basalis which reacts by infiltration and thickening (see p. 1724), but also into the bordering myometrium. This occurs contrary to the physiological conditions (p. 1716); in cases such as that of Marchand, it takes place to such excessive degrees that the line of differentiation between the compact and cavernous layers of the deciduae is entirely destroyed; the so-called decidual septa are lacking; many efferent and afferent vessels are obstructed, while other vessels are opened by the vigorous cell growth. This, in contradistinction to that of the normal, is of much longer duration and of much greater intensity and extent, although these characteristics are not so outspoken in every case (Langhans-Chaletzky).

With these remarks in mind, it is easy to understand the conditions in the so-called interstitial destructive hydatidiform mole such as described by v. Volkmann (see also Kauffmann). The decidua is broken through by the vesicular villi which grow into the blood vessels of the decidua basalis, spread themselves in the veins of the uterine musculature and reach under the peritoneum, or even into the parametrium. They have been found in dilated veins (see Amreich), and have even partially obstructed the ureters (see Amann). Perforation, peritonitis, internal hemorrhage (grave danger in curettage, see v. Franque), and metastases may follow. Dunger believes this structure occupies a position between hydatidiform mole and chorion epithelioma. Curtis and Oui (lit.), who in general consider all these destructive hydatidiform moles malignant, have described a case in which the growth was completely circumscribed, purely local, and relatively benign. Marchand's observations help one to understand the cases in which the hydatidiform mole is malignant, but its malignancy must be determined not from single isolated villi, but by a consideration of its destructive activity in the neighborhood (see p. 1745). We can also understand those cases in which destructive epithelial growth arises from rests of a hydatidiform mole remaining in the region of the decidua basalis. According to Seitz, this occurs in about 6½% of the cases; according to Sunde it is more frequent in women over 40 years of age. It is a short step between the vigorous cell overgrowth of a hydatidiform mole to a malignant tumor. There occur wide spread cellular transports into the lung in hydatidiform mole, which, of course, are without consequences, and which, according to Dunder, heal through the agency of connective tissue increase. These various characteristics have led to the idea that the hydatidiform mole is a structure on the border line between malformations and tumors, (see also Broman). The malignant chorion epitheliomas (L. Pick, Marchand) will be discussed in more detail on p. 1739 (lit., Risel, see also Daels).

Placenta marginata which has been described on page 1716, as well as placenta circumvallata, in which the rim of the placenta is bent over, or as others depict, is lifted up by fibrin deposits between the leaves of the reflexa, is of great obstetrical interest. (Lit. and theories concerning the origin by Liepmann, Sfameni, and particularly, R. Meyer.)

Diseases of the Placenta. Regressive Changes.—Calcification takes place in every mature placenta and involves the fibrin masses particularly (Merttens). Fatty degeneration occurs in the decidual parts of the placenta. It may be manifest as white spots.

In nephritis of the mother, which may be accompanied by nephritis in the fetus, (see Sitzenfrey), edema of the placenta, together with hydrops of the fetus, is quite common.

Fibrin plugs or thromboses of the maternal blood spaces, the so-called white infarcts of the placenta are opaque masses, red, light gray or yellowish white in color, and are recognized by rounded or irregular nodules or wedges, or flat places (fig. 870). On the edges of the placenta they

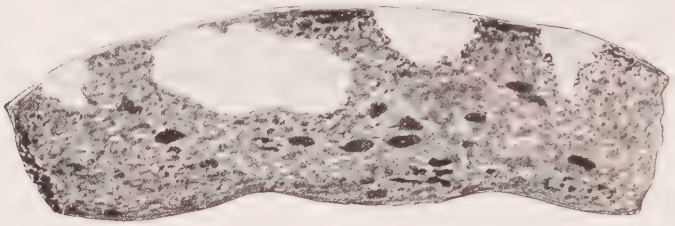


FIG. 870.—Multiple thrombi, so-called white infarcts of placenta; nat. size.

appear as a tough ring raised above the level, or they involve larger areas which are often sunken. At times most of the placenta, indeed even the entire placenta, (unilateral in twins) may be involved. The central portions of many of the wedges of larger size may be softened to a whitish red or yellow pulp (almost suppurative).

FIG. 871.—So-called infarct of placenta; a, normal villi, surrounded by blood; b, longitudinally cut villus undergoing necrosis; in center of illust. an intervillous fibrinous thrombus; inside of it the villus b, and two other necrotic villi cut transversely; epithelium lost where villi border on the thrombus. (Med. power.)

Microscopically, there appear necrotic masses in which the intervillous blood spaces are obstructed by fibrinous thrombi; in the fibrin may be enclosed fetal or decidual tissue, or both. Red and white blood cells, or blood pigments, may be present here and there. Older masses contain calcium at times. The fibrin is homogeneous, traversed by spaces

(canalised fibrin, Langhans), or finely fibrillar; in the softened puriform areas, it is a finely granular mass of detritus. The epithelial cells of the villi disappear inside the thrombus or where they touch the thrombus; the stroma of the villi undergoes hyalin degeneration, the nuclei cannot be stained, but are cloudy or glassy, vessels are completely obstructed, or may not be recognizable (fig. 871). In the neighboring decidual tissue there may be no changes, or perhaps overgrowth which implies beginning organization of the entire region, but, on the other hand, this latter change may be primary, and may have led to the formation of the infarct. In the latter case, there is a primary "decidual endometritis." The decidual supporting substance may also be hyalinized here and there, or show stripes of fibrin and necrotic cells.

Small infarcts are frequent, but probably not pathological; the child may be entirely healthy and vigorous. Numerous larger thromboses lead to destruction of larger vessels, and thus the fetus is poorly developed or is killed. If the serotina is diffusely involved, loosening of the placenta may be difficult.

Etiologically, endometritis and nephritis must be considered (Fehling, see also Seitz, Kworostansky). Definite evidence of syphilis cannot be found anatomically, but there seems to be a relatively greater frequency of these sub-decidual thromboses when the mother has syphilis.

It may be remarked that fibrin is found in the normal placenta from the second month onward and toward the end of pregnancy, on the placental surface of the chorion (Langhans), where it appears on the mature placenta as gray spots, on the placental side of the serotina where the fetal and the maternal deciduae touch (Nitabuch's fibrin stripes), and on the surface of the villi where the syncytium is lacking. (The syncytium is said to have anticoagulative powers, Eberhardt.) This fibrin, as well as everything which has been called hyalin in the placenta, takes Weigert's fibrin stain in the early stages, but not later (Merttens). The source may be degeneration of the chorionic epithelium or decidual cells, or secretion or excretion on the part of the chorionic cells, or thrombotic coagulations from the blood.

Views Concerning the Origin of the So-called White Infants.—The same diversity of opinions as expressed concerning the normal fibrin in the placenta, appears concerning the origin of the fibrinous wedges which are so often found, and which are probably not even pathological. Of these views, a few may be mentioned: The primary changes are said to be: (*a*) changes in the deciduae; decidual cells overgrowing as a result of endometritis surround the villi and later undergo hyalin degeneration (Steffeck; see also Baldowsky). (*b*) Disease of the vessels in the stalks of the fetal villi, but not as Ackermann previously assumed in the form

of a periarteritis nodosa, but in the form of an endarteritis (Favre, Ackermann), which leads to ischemic necrosis of the villi and subsequent thrombus formation in the intervillous spaces, *i.e.*, to an anemic infarct. (These vessel changes are considered by many, but erroneously so, to be due to syphilis.) Later, Ackermann assumed that the process was not one of thrombosis, but of secretion or degeneration of the chorionic epithelial or decidual cells. Any relation of the entirely inconstant arteritis to the origin of the fibrin wedges is very doubtful. The findings of Merttens are of interest in this connection. In examinations of placentas which had remained for some length of time in the uterus after death of the fetus, but which were in good nutritional condition, he found there was an overgrowth of the intima, particularly in the arteries in the stalks of the chorionic villi and this led to obstruction; but in the "infarcts" which were found in such placentas, these vessel changes were not found. These changes are considered by many to be signs of syphilis (Prinzing), but since syphilis was eliminated in the cases of Merttens, they can hardly be characteristic of the disease. (*c*) Many authors have recently adopted the opinion that there is a primary hematogenous thrombosis in the intervillous spaces. It is not improbable that the beginning of coagulation is dependent upon destruction of the chorionic epithelium which has anticoagulative powers (Eberhardt). Hitschmann and Lindenthal believe that these infarcts are due to a particular kind of thrombosis from slowing of the blood stream and changes in the epithelial cells. Secondary regressive changes produce the later characteristic appearances. The author has believed in this idea of thrombosis and fibrin wedges for many years (see the second edition, 1901). Schickele proposes the name "fibrin nodules." Clemenz speaks of "white necrosis." (*d*) Kalima (lit.) calls attention to ectodermic overgrowths in addition to the primary thrombosis; the former, he thinks, is often primary, and leads to hyalin degeneration of the villi; neighboring parts undergo liquefaction and the blood lying in between stagnates; secondarily, fibrin is laid down, partly as blood fibrin and partly from hyalin degeneration of the fetal parts of the placenta.

Infectious Granulation Tumors of the Placenta.— It is often difficult to testify that syphilis has produced changes in a placenta, and in general, it is not always possible, on the basis of histological examination of the placenta alone, to make the diagnosis of syphilis in the fetus. There are, however, cases in which certain changes are found from which a definite conclusion may be drawn. It is always to be recommended, however, when possible, to examine not only the living or dead child (see p. 1133) but also the umbilical cord (p. 1734), and to take these results into consideration with the placenta before an opinion is given. Often, the

placenta of a poorly developed child is large, plump, heavy and perhaps tougher and paler than normal.

The normal placenta of a mature child weighs somewhat over 500 grams. Its thickness is 2.5 to 3 cm., its length 16 to 20 cm. It is as 1 to 6 in relation to the body weight; in syphilis the relation may be 1 to 3 (Rosinski, Mracek). Chorionic hyperplasia and edema produce the increase in weight, volume and consistency.

The microscopic findings are not constant. Ernst Frankel described a "placentitis fetalis," in which there are a nodular swelling and a macroscopic plump thickening of the chorionic villi; he called the condition "deforming granulation cell swelling of the placental villi;" the stalks of the villi are involved in a cellular proliferation; at the same time, the villous epithelium also undergoes proliferation. These findings were confirmed in part, but in part were denied (lit. by Thomsen). Rosinski also described a diffuse inflammation with universal proliferation of the tissues and even hyperplasia of the stroma, the vessels and the epithelial covering. The process is said to originate in the smallest vessels. But this emphasis on a vascular origin is too decided, and is not applicable to many cases. That there is a cellular hyperplasia frequently combined with edema of the villi can be confirmed by the author, and he believes with Thomsen (lit.), that a severe degree of these changes is seen only in syphilis (see also Schmidt-Baumler). At the same time, there is often leucocytic infiltration in the fetal membranes which may even lead to the formation of abscesses. The maternal parts of the placenta are often found free of characteristic or specific changes (Thomsen). Mracek (lit.) believes that changes in the vessels are more important. The vessels are said to be nodular, rigid, tough-walled, with narrowing of their lumena, even to complete obliteration. Schwab, who also admits the presence of a large cell villous overgrowth, describes endo- and periarteritis and phlebitis (see also Franceschini). Even the formation of gummas, "gummatous placental endometritis," involving particularly the uterine surface of the placenta, has been described (Virchow, E. Frankel). But these may have been only the so-called infarcts, which may or may not be more frequent in syphilis. Calcified areas may also be particularly numerous, especially in the stroma and in the vessel walls of the villi; the latter may also undergo fatty degeneration. Hitschmann and Volk deny that any or all of these changes as described are specific of placental syphilis, but we must differentiate between "specific" in the strict histological sense, and in the "pathognomonic" sense. The cellular hyperplasia of the villi is probably pathognomonic of syphilis.

By the use of Levaditi's silver method, Paaschen, Wallich and Levaditi and many others have found siprochetae particularly in the villi; Mohn,

e.g., found them in 50% of the cases in the umbilical cord, and in the placenta, in 70% of the cases of syphilis of the parents, (see also Bab). According to Seitz, Trinchese found spirochetae in all cases. Massone states that even though the reaction of the placental blood to the Wassermann is plus four, the maternal blood may be negative. (Illustrations of spirochatae on p. 1286.) See also under umbilical cord, p. 1734.

Involvement of the placenta is not uncommon in acute miliary or uterine **tuberculosis,** or in severe pulmonary tuberculosis of the mother. (Literature in appendix.)

Eventually, all the parts participating in the formation of the placenta may be infected from the mother, usually by the hematogenous route, but sometimes by direct extension from a genital tuberculosis (Schmorl and Geipel). The most frequent forms are bacteria-containing caseous masses between the villi in the maternal sinus; the villi themselves may be intact or also involved. But the villi may be independently involved, in which case the bacilli reach their covering through spaces and establish themselves in the connective tissue layer and in the vessels, and develop typical tubercles. But actual involvement of the villi may be lacking in spite of many defects in their epithelium and much tuberculosis in the intervillous spaces (Lanz). The fetus may be infected; according to Sitzenfrey this placental involvement by tuberculosis seems to be much more frequent than was formerly assumed. It may occur without changes in the placental tissues, when, for instance, during childbirth, damage to the vessels of the villi allows the entrance of bacilli into the intervillous spaces (see Leuenberger and also Novak and Kanzel). Rapidly caseating round cell infiltration, and typical tuberculous masses (Sitzenfrey) may be found in the decidua basalis as well as in the chorionic covering of the placenta. This occurs by rupture of Nitabuch's fibrin stripes, entrance into the intervillous spaces, villous tuberculosis, destruction of the attachment of the villi, and following this, hemorrhages and abortion. The decidua vera may also be involved in this diffuse caseating form without involvement of the placenta (Schrumpf). See Lanz, who observed miliary tuberculosis of the decidua vera.

Invasion through the placental filter from the mother to the fetus has been demonstrated for many other microorganisms. There may be mentioned: gas gangrene bacilli, anthrax bacilli, tetanus bacilli, spirochetae of recurrent fever and of syphilis, the typhoid organisms, pneumococci, staphylococci. But, on the other hand, entrance to the fetus may not take place. The conditions for invasion into the fetal circulation are sub partu (Gartner) particularly favorable, and changes in the placenta, particularly hemorrhage, favor it (Malvoz, Birch-Hirschfeld and others).

For the filtration of toxins from the mother to the fetus, see, among others, Schmidlechner.

Tumors of the placenta are rare; usually they come from broken off nodules or by direct extension of fibromas, myxomas, angiomas (Storch, Niebergall, Bode and Schmorl, Albert, Beneke and others). Dienst (lit.) is of the opinion that all placental tumors are "allantogenous-myxo-fibro-capillary angiomas of the chorion," and according to the relative amount of blood vessel or connective tissue, they may be called "chorioma angiomatosum" or "fibrosum." The "allantogenous" origin (Dienst) has, however, not been demonstrated (Schickele). Solowij believes the so-called placental tumors are products of a placentitis, particularly syphilitic. Kraus, Seitz (lit.), Pitha (60 cases from the literature and 3 new ones), Kermauner, Ravano and others believe, on the contrary, that these growths are of true tumor nature (angiomas). Schindler considers stasis (through kinks?) a cause of the growths. Grafenberg again denies that they are tumors, while many others believe in their neoplastic nature. Bohi speaks only of malformations. On the basis of four cases, the author feels that there do occur capillary angiomas of the villi, that the connective tissue may be richly cellular like the embryonal connective tissue, or fibrous or mucoid, and occasionally overgrown (in which case it is proper to speak of fibro-angiomas). In these four cases, the tumors were as large as cherries, hen's eggs, and apples, were tough, more or less lobulated and nodular, sharply outlined, connected to the chorion by vessels in pedicles, and loosely attached to the remainder of the placental tissue. See also Eicke, Hauser, Kummer, Elten, Joetten, Johnstone, Nebesky, Ribbert, Kohler, and the work from the pathological institute of Gottingen by Roscher, Williams, Acconci.

Subchorial cysts of the placenta occasionally reach the size of cherries, and are of doubtful origin. According to Albeck and E. Runge (lit.), they are softened overgrown decidual cells which arise under the chorionic membrane and thus push forward in hemispherical shape into the cavity of the ovum. Blau (lit.), on the other hand, accredits their origin to overgrowth and degeneration of fetal cells arising from the trophoblastic Langhans cells (Brohm, Biland), as well as to hemorrhages and liquefaction of infarcts (see also Baldowsky).

Secondary tumors of the placenta. The case of Senge (lit.) is unique: metastases in the placenta from a carcinoma of the stomach. Walz and Marcus have described metastases from a sarcoma.

Changes in the Umbilical Cord. Uncommon Insertions.—Instead of a central insertion, there is seen occasionally a marginal insertion, a velamentous insertion (see v. Franque), or a forked insertion of the umbilical vessels. True knots develop when the embryo slips through a crossed

open loop; this occasionally leads to death of the fetus. False knots are collections of Wharton's jelly around convoluted and tortuous vessels. According to Thoma, they arise from hindrance to the usual spiral windings of the blood vessels. The physiological spiral windings are from turning of the body of the fetus in the amniotic cavity; pathological torsions occur in the same way. The normal length of the umbilical cord is about 50 cm., and about equal to the length of a normal child. The stump of the umbilical cord falls off, usually on the fifth to the sixth day, following demarcation at the base of the umbilical cord. Abnormal length, even up to 190 cm., abnormally numerous and tight windings, turnings in the long axis (torsio nimia), may occur from movements of the living, or, as is stated, from passive movements of the dead child. The windings of the first mentioned condition are replaced with difficulty; their form is permanent. A fetus with torsio nimia is often born prematurely (in the fourth to the fifth month) and may be very atrophic. Twists of a long umbilical cord are quite frequent. Occasionally this is a source of grave danger to the fetus, *e.g.*, when it is wound around the neck several times. Pressure on vessels and nerves may result, *e.g.*, in atrophy of an arm. The twisting together of both umbilical cords in a one ovum twin (communal amnion) is rare, (see Piltz, lit.); death of one or both embryos may occur. See Kusumi, lit. concerning the lack of one umbilical artery.

In syphilis of the child or the mother or of both—although it occurs in but half the cases, (Simmonds, lit.)—there appears an inflammatory infiltration involving the stroma of the cord and particularly the vessels (2 arteries, 1 vein), consisting chiefly of leucocytic infiltration of the walls (media and intima), which become thicker because of this vasculitis. But the author has also seen cases with exquisite productive endarteritis and thrombosis. Small vessel gummas also occur (Dominici, lit.), but some of these cases may be mistaken for ordinary necroses; the latter are infiltrated by polymorphonuclear cells and perhaps calcium granules. The syphilitic changes vary much in intensity and usually involve only parts of the umbilical cord; they are without specific histological characteristics, but since other infections coming from the placenta play only a small role in their development, (Simmonds found them in 9%, Creadick in 2% of nonsyphilitics, see also Rietz), they therefore have some significance. Bondi (lit.), however, goes too far, as Simmonds and Creadick point out, when he believes that these umbilical cord inflammations are pathognomonic of lues, because they may be absent in undoubted cases of syphilis, and in other cases may be present when there is no syphilis. The view of Thomsen is easier to accept, namely, that every child, whose parents are syphilitic, is also diseased if it shows these inflammatory changes in the umbilical cord (see also Schmidt-Baumler). The demon-

stration of the spirochetae pallida is, of course, conclusive; Grafenberg found them most frequently in the fetal parts of the umbilical cord; Dominici (lit.) found them in almost every case. The ends of the umbilical cord, which is usually thickened as a whole, are, as a rule, most frequently and intensively diseased; according to Bandi the fetal, according to Dominici, contrariwise, the placental end, is more affected; the vessels are not always evenly involved. See Manouelian concerning primary phlebitis of the umbilical vein and its relationship to infection by the spirochetae. Thrombosis may follow. Irregular, simple thickening of the intima of the vessels is not characteristic, but occurs under normal conditions. In the umbilical vein of a macerated fetus, there are often fatty degeneration and calcification of the intima. Sitzenfrey has described tuberculous changes in the umbilical vessels.

New Growths are Very Rare.—In a new born, the author described a telangiectatic myxosarcoma of the cord the size of a small apple; it appeared a short distance away from the navel. Herweg (lit.) has reported an analogous case which the author has also seen.

R. Meyer described a teratoma of the umbilical cord in a new born. Oberndorfer writes of a tumor larger than a man's head in the round ligament of the liver (endothelioma sarcinatides), in a 24 year old woman. He believes it arose from the canal of the umbilical vein. Chiari described a partial defect of the intraabdominal portion of the umbilical vein with the formation of varices.

See p. 735 for umbilical hernias; umbilical adenomas are described on p. 726.

See Reifferscheid concerning tearing of the umbilical cord and separation at the navel by precipitate labor.

Changes in the Ovum and Fetus after Intrauterine Death. Abortion. Blood or Thrombus Mole.—These follow the death of the fetus in the first few weeks; successive hemorrhages separate the layers of the deciduae and gradually loosen the products of abortion; when the blood clots are expelled, these structures appear. The chorion and the amnion, because of their deciduae which bring nourishment by osmosis, remain intact for a certain length of time. Hemorrhages take place in the serotina, partly loosen the vera, and force themselves between the chorion and reflexa. According to Neumann, they force the chorion and amnion against the egg cavity like a tumor (tuberous subchorionic hematoma), in which, according to Breus, there is first a diverticulum-like lifting off of the chorion and amnion, and then hemorrhage, which may even enter the cavity of the ovum. According to Endelmann (lit.), these are not actual hematomas, but only blood coagulation in the intervillous spaces. Compare Waldstein (lit.) who calls this mole a "hematoma mole" or an "aneu-

rysmal ovum," and the fleshy mole, an ovum saturated with blood (missed abortion). On section through a blood mole, the amniotic cavity is covered on the inside with amnion and outside with villi, and surrounded by a thick covering of layers of blood coagula; the embryo is absorbed or may be present in microscopic traces. If death occurs in the second to the third month, there usually remains a softened fetus (see also Greil). If the blood mole is decolorized from a long sojourn in the uterus, the mole is lighter colored brown or yellow red, tough and fleshy. After a time, calcification of a fleshy mole produces the so-called stone mole (rare). The epithelial covering of the villi (trophoblast) may be remarkably well preserved, as seen, *e.g.*, by Trancu-Rainer in a mole retained in the uterus about eleven months. When the fetus dies in the later months of pregnancy, from syphilis of the mother or obstruction, or interruption of the placental circulation, and when it remains in the uterus for a time, all parts are soft, macerated, and liquefied to a finely granular detritus; the subcutaneous tissues of the fetus are spongy and soaked with diffuse blood pigment and amniotic fluid (fetus sanguinolentus); formerly it was erroneously called "decomposed fetus." The skin macerates, the brain liquefies into a pulp, the bones of the head hang loosely. The serous cavities contain red fluid. The blood is strikingly changed by hemolysis; it is thin, fluid, cloudy brown red, and usually contains numerous crystalline bilirubin precipitates. The liberation of fatty acids in the connective tissues of the organs leaves fat crystals; the organ surfaces, particularly of the liver, show yellow precipitates. The amniotic fluid is cloudy; it has a sweet, elusive odor. Decomposition does not take place in the closed amniotic cavity; a stinking decomposition occurs only when the bacteria of decomposition reach the fetus from the vagina. A dead fetus may become mummified; this is occasionally seen in twins, and sometimes a living child is born with a mummified twin. Sometimes the mummified fetus is flattened (compressed fetus), or as thin as paper (fetus papyraceus).

By abortion is understood the early interruption of pregnancy before the fetus is viable; in premature birth, on the other hand, the fetus is viable (from the 28th week onward). In the former, the masses expelled are called abortivei.

In the first two months, the abortion consists principally of maternal membranes. As stated (p. 1719), a three cornered sac containing the ovum is expelled. From the third month onward, the ovum comprises the chief mass, and the condition of the placenta now becomes of importance. The ovum is expelled into to, or if torn, the fetus slips through the amnion, and the amnion and the placenta follow. Infrequently, the sac is expelled first and the chorion and placenta remain for a time in the uterus.

According to Schottlander, in the first to the third month of pregnancy, nucleated red blood corpuscles in gradually decreasing numbers are found in the vessels of the villi and chorionic membranes; the stroma is poor in cells.

In infectious abortion, the fetus is infected in only a part of the cases (for more details, see W. Barfurth).

It often occurs that portions of the maternal and fetal membranes remain behind, and this is spoken of as incomplete abortion. Portions of the deciduae which are more in evidence during the first two months, may be spontaneously expelled over a period of weeks. In the later stages of pregnancy, chorionic parts or large pieces of the placenta remain behind; hemorrhages occur, the uterus is curetted and the material subjected to microscopic examination.

It must be remembered that even in the absence of formed villi which remain in the decidua basalis, fetal cells are demonstrable for months, especially those which had wandered into the mucosa from the surface of the villi in the region of the placenta (p. 1716). These cells are different from the other tissue cells and are of variable size, rounded or angular, spindle or oval, and occasionally polynuclear. They are characterized by dark, chromatin-rich, polymorphous nuclei and are differentiated quite easily from decidual cells. They are signs of a previous pregnancy (Aschoff-Gaylord). Schickele lays particular stress upon the finding of such large cells in the walls of hyalinized vessels, especially arteries, in which they are preserved the longest. See also placental polyps (p. 1738).

In the differential diagnosis there should be considered: (*a*) membranous dysmenorrhea (p. 1620); but this occurs, and membranes are expelled, every four weeks. (*b*) Expulsion of the deciduae in an extrauterine pregnancy from separation in the spongiosa by leucocytic infiltration; the differentiation of these so-called ectopic deciduae from true deciduae of uterine pregnancy cannot be definitely made from the expelled particles (see also Lahm). Even in seemingly smooth and empty sacs which are taken to indicate extrauterine pregnancy, great care must be exercised in searching for a small nodular thickening at some situation in the internal surface in which the ovum may be hidden. (See also pp. 1621 and 1709.)

The diagnosis of abortion was thought possible by Opitz, by finding markedly overgrown, wide, dilated glands showing papilla-like or basket-like epithelial buds growing closely together into the lumen in larger portions of the mucous membrane. The stroma would therefore be pressed together and occupy a small space, such as is normal in the spongiosa (see p. 1710); thus other areas similar to the compact layer are brought into sharp contrast. The epithelial overgrowths may simulate

the picture of a papillary adenoma. Probably this condition is a reactive regenerative process in the mucous membrane which oversteps the mark, so that the changes may be termed "hyperplastic endometritis after abortion" (see Kustner); see p. 1622. But opinions are divided concerning the diagnostic importance of these papillary glandular forms, since the changes occur without pregnancy, even though rarely; thus, they are seen in endometritis (Seitz), in premenstrual swellings (Hitschmann), and, on the other hand, may be absent in true abortions. The author has often seen these pictures under the circumstances outlined by Opitz; on the other hand, he has seen them in most typical form in nodular masses coming from the uterus at the end of pregnancy, the uterus having been emptied manually because of hemorrhages. The author therefore cannot believe that they are a particular or a characteristic diagnostic sign of abortion or of an early stage of pregnancy; these glandular pictures are not at all pathognomonic of pregnancy, since they occur under certain irritative conditions outside of pregnancy (see also Ruge). Very frequently, after abortion, the author saw groups of richly cellular small arteries (6–12) in the mucosa, (curettings).

6. The So-called Fibrinous or Placental Polyps. Hyperplasias and Malignant Tumors, Which Arise from Placental Rests or Parts.—After an abortion or a normal childbirth, remnants of the membranes, but more frequently of the placenta, which are connected with the maternal tissues, may remain weeks or months in the uterus; they hinder regeneration of the mucous membrane and disturb the involution of the musculature; hemorrhages are frequent accompaniments. Blood may cover these rests like shells and round them off (figs. 872 and 873), and the successive addition of masses of coagulated blood produces polypoid tumors as large as fists. Since, as a rule, they have a nucleus of placental tissue, they are called "simple placental polyps," or, according to the most prominent of the constituents, "fibrin polyps" or "polypoid uterine hematomas." (Schickele also emphasizes their varied structure.) Chorionic epithelial masses may be preserved for a long time within these structures; they at times show a vigorous growth, which leads to invasion of the maternal tissues (uterine walls). At first, this is a benign chorionic epithelial cell invasion, but the possibility of a malignant tumor is always present. For clinical purposes a microscopic examination must always be made (see also Fink). As a rule, however, if they are not removed by operation, they are expelled spontaneously, sometimes after previous gangrenous destruction. The case of Zahn is unique; after a probable duration of thirteen years, a simple benign placental polyp of the anterior wall led to pressure necrosis of the opposite posterior wall of the uterus which finally perforated and produced a retrouterine hematocele.

If the reparative processes in the mucous membrane step over the mark after child birth or after abortion, particularly the latter, or after removal of a hydatidiform mole, there may arise a diffuse or a polypoid hyperplasia of the mucous membrane. Islands of decidual tissue and even remnants of villi may be enclosed in these growths (Sanger, Marchand). See pp. 1622 and 1738 concerning the hyperplasias which follow incomplete involution of the uterus in early abortions.

The most important of the malignant tumors which follow abortion or normal child birth, but which often arise during pregnancy as well, in the

FIG. 872. FIG. 873.

FIG. 872.—Placental polyp inserted into the right uterine cornua of a 30 year old phthisical woman; fresh, it was pale gray yellow; microscopically, polyp consisted chiefly of fibrin (fibrin polyp), containing necrotic chorionic villi here and there with blood in between; 5/6 nat. size.

FIG. 873.—Longitudinal section of polyp with uterine wall into which it is inserted; 5/6 nat. size.

uterus, or, in rare cases, in the pregnant tube, are called **chorion epitheliomas.** They originate from the cell covering of the placental chorionic villi. (See p. 1600; lit. by Risel, Hormann, Sunde.)

They appear at the situation of the placenta, *i.e.*, in the region of the decidua serotina, and in almost one half of the cases (according to Seitz in 40%) are preceded by hydatidiform moles (p. 1727). The tumors are irregular and villous and resemble simple placental polyps on the surface, or are flat and spread out on the inner surface of the uterus (fig. 874), of red or dark brown color, mixed with white tissue and necrotic areas; the

metastases are frequently infiltrated with blood. Those arising from the situation of the placenta grow in the midst of much hemorrhage into the musculature and metastasize via the blood stream similar to sarcomas; metastases have been found in the lungs, vagina, brain, liver, kidneys, ovaries, etc., and also in the regional lymph nodes. Sanger called the tumor "sarcoma deciduo-cellulare" and believed that the decidual tissue was the point of origin. L. Fraenkel and particularly Marchand, however, showed that it was an epithelial new growth. In contrast to car-

FIG. 874.—Malignant chorionepithelioma of uterus (⅘ nat. size). Uterus cut open anteriorly in median, and above in transverse line, shows in upper portion a broad-based polypoid tumor lamellated on section which also penetrates the loose parietal layers; woman 31 years old; almost continuous hemorrhages for 3 months; metatases numerous in both lungs (fig. 215); expectoration bloody. The l. kidney contained a nodule larger than a hen's egg, and from there a tumor plug as thick as a lead pencil continued into renal vein. Both ovaries cystic; one nodule each in l. ovary, spleen and brain (fig. 952). All nodules multicolored and contained blood lacunae (consult the author in Corresp. bl. f. Schweizer Aerzte, 1900, 10).

cinomas, which show an organoid structure, they consist only of epithelial cells. Marchand was first of the opinion that the two chief cell forms which appear in the tumor, namely, cell syncytia or large cells which in their compact protoplasm, their nuclei and so on, resemble the syncytium, and secondly, the light polyhedral cells of the type of the Langhans layer, arose from two sources, the former from maternal, the latter from fetal tissues. After more data concerning the embryology had accumulated,

this view was no longer tenable, and later, Marchand believed with others that just as both layers which cover the chorionic villi most probably come from fetal ectoblast (see p. 1712), so also do the cellular components which occur, as a rule, in the malignant chorion epitheliomas. Both cell types may be along side of each other or be mixed together (fig. 877); the "typical form" consists of a tumor composed of joined, irregular, many-nucleated branching sycytial masses with fine fat droplets, and lighter, small, polyhedral, glycogen-containing cells of the Langhans type with many

FIG. 875.—Typical malignant chorionepithelioma; from a metastasis in the mesentery; a, Langhan's cells; b, syncytial bands; c, thrombi composed of platelets, fibrin and leucocytes; d, blood. (Med. power.)

nuclear divisions (Marchand). The latter appear (as in fig. 877) as masses of light staining, glycogen-rich, polyhedral cells, separated and surrounded by more or less broad bands and septa of intensively staining syncytial masses. In the "atypical forms" which are much rarer, the peculiar trabeculated arrangement of the chorionic epithelium is found only in places, or perhaps not at all; isolated cells alone are seen, compact, strongly staining and very irregularly formed, often with giant nuclei and similar to giant cells, but nevertheless somewhat resembling the syncytium, the chorionic wandering cells and true serotinal giant cells. On the other hand, this lively mixture also contains cells which resemble those of the

type of Langhans cells. The tumor masses may infiltrate the uterus more or less diffusely. There are, however, appearances reminiscent of the physiological chorionic cell invasion (p. 1716), even infiltration into the vessel walls; but everything takes place to a colossal degree. Confusion with the giant cell sarcoma, (therefore the name, "sarcoma deciduo cellulare," Sanger), is quite easy, and Veit pleaded, of course without results, for the sarcomatous nature of this tumor; finally he also agreed to the opinion of Marchand. Others called the tumor "malignant decid-

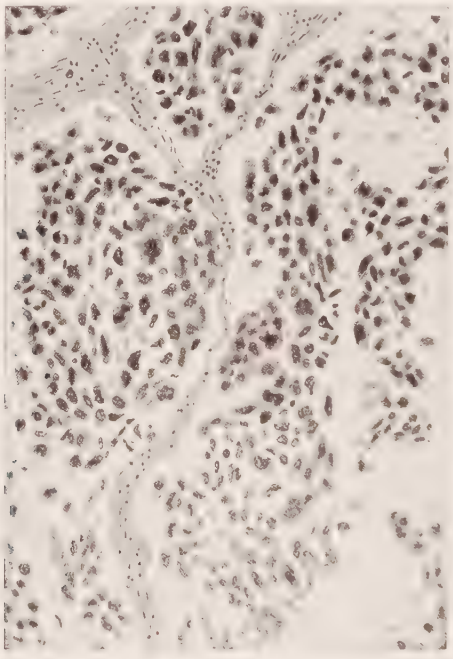


Fig. 876.—Atypical malignant chorionepithelioma infiltrating the wall of the much enlarged uterus; syncytium has separated in individual cells of marked polymorphy; woman 35 years old. (Med. magn.)

uoma" (according to Winkler); but it has no relationship to the deciduae. Others, who believe in the chorionic epithelial origin, use the terminology "syncytioma malignum;" others speak of carcinoma of the uterine body as of placental origin (Jayle and Halperine); but Marchand's nomenclature seems to be preferable (see also Sunde, Nagy and others). There are transition forms between the typical and the atypical types; the atypical types are usually more malignant, (Heimann).

The close relationship between malignant chorion epitheliomas and hydatidiform moles has been emphasized on p. 1727. The condition seems to be merely an increase of the lawless chorionic epithelial overgrowth, which is seen quite plainly in a hydatidiform mole, and is still more

pronounced in the destructive form of the mole. Even in normal pregnancy there is an ingrowth of villi into open uterine veins and an overwhelming of the upper mucous layers with infiltrations, not only of the surface muscle bundles, but also of the deeper layers; there is emigration of villi and loosened villous epithelium into the vessels of the lungs, (see p. 1716), but this overgrowth is limited and the embolic cells, as a rule, are soon destroyed (for the exception, see p. 1738). In malignant chorion epithelioma, on the other hand, there are an extensive infiltration of the wall of the uterus by excessive cell overgrowth and the production of

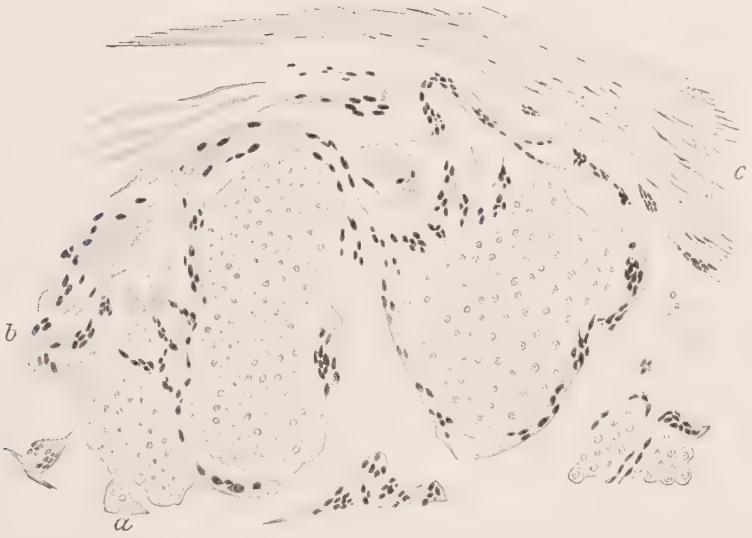


FIG. 877.—Malignant chorionepithelioma of uterus, typical form, developed after hydatidiform mole; contents of a uterine vein; a, cells of type of Langhan's; b, syncytial cells staining very well; c, myometrium. Ten weeks before a hydatidiform mole removed; since then continued hemorrhages; pronounced anemia. (Med. magn.)

numerous rough growths into veins. The cells of the tumor have an extraordinary growth energy, and reproduce independently and quickly when they are carried onward. It is interesting that here and there in the malignant tumor is actual formation of villi with wide meshed edematous stroma as a reminder of the hydatidiform mole (*e.g.*, in the case of Aschoff-Apfelstedt); such a condition may be spoken of as "malignant hydatidiform mole;" metastases may even occur during the pregnancy; see, for instance, the rare case of Gustafsson (lit.) in which in the midst of a normal pregnancy (embryo 4 cm. long), a chorion epithelioma of the uterus arose, with metastases in the vagina and lungs. In other cases there is a latent period which may last many years (1 13) between the hydatidiform mole, *i.e.*, pregnancy, and a manifest chorion epithelioma (lit. by Krosing and Polano; see also lit. of Fischer). Sunde (lit.) found chorion epitheli-

oma occurring after hydatidiform mole much more frequently in women past 40 years of age.

The relationship of the tumor to the veins is particularly intimate; this explains the sarcomatous mode of spread with early and numerous hematogenous metastases, particularly in the lungs. This also explains its hemorrhagic appearance and the formation of cavernous blood spaces, formed lacunae, and the mixture of fresh and old thrombi and blood coagula which also occur in the metastases (figs. 215 and 952). The chorion epithelial cells often lodge around the veins and then rupture into them. Other veins are completely filled. There then occur thromboses, bloody infiltrations of the neighborhood with fibrin masses, and necrosis of the tissue, stasis over wide regions, and all of these conditions help give to the tumor its peculiar hemorrhagic character and the softneed yellow red and brown speckled appearance of the section surface.

Among the metastases are those in the vaginal wall from retrograde transport in the vaginal veins. These are the commonest; next are the metastases in the lungs, which often lead to hemoptysis (see Fig. 215) and the metastases in the liver and brain (see fig. 952 under brain). But metastases have been present in many other organs, such as the kidneys, spleen, etc. (Schottlander.) They sometimes show prominence of the syncytial masses or perhaps large cell masses which resemble sarcomas; in other tumors the cells have the characteristics of the syncytium. Occasionally they have a peculiar latent period. Thus, Koritschoner describes metastases appearing 22 years after the removal of the primary tumor in the uterus.

Schmorl described a primary chorion epithelioma of the vagina manifested 18 weeks after the end of a normal pregnancy. Vaginal nodules may appear not only in the malignant but also in the benign hydatidiform mole of the uterus, and even after simple abortion (L. Pick, and under vagina, p. 1698). There are also numerous observations in the literature of ectopic malignant chorion epitheliomas during and after pregnancy, in situations other than the region of the insertion of the ovum (lit. by Zagorjansky-Kissel, Hubl, Risel, newer literature by Fischer). According to Schmorl and Dunger, there is probably a change of the ovum or of the placenta in the manner of a hydatidiform mole, since the appearance of the proliferations of the emigrated elements are those of a mole and not those of a healthy placenta. It is worth while remembering that hydatidiform mole formation may take place in isolated villi and may be overlooked. It is also permissible to assume that deported harmless materials (cells, villi) may become malignant at the situation where they are stopped (see also Nagy); Walthard described a case in which, in the second half of pregnancy without a primary tumor in the uterus, a vaginal

tumor and a generalized chorion epitheliomatosis occurred from cell embolism. In spite of the most careful microscopic examination of the placenta, nothing pathological was found; of course, the objection of Veit is pertinent, namely, that all parts of the uterus showing hydatidiform mole had already been embolized. In a remarkable case described by Busse, tumor formation took place as a parietal thrombus in the heart, from which emboli arose and were lodged, particularly in the brain, but also in many other organs. In other cases, the heterotopic chorion epithelioma following pregnancy was found primarily in the liver (Gurewitsch), in the ovaries (Klotz, Risel). In a 25 year old woman who had given birth to her second child five months previously, the author saw an enlarged but otherwise unchanged uterus; there were many nodules in the lungs with rupture into veins, numerous rounded, blood red masses in the brain (the woman had convulsions, paralysis of the extremities and speech, and unconsciousness), infarcts in the spleen and kidneys with tumor thrombi in the arteries (compare cases of Eichhorn). See also literature by Woenckhaus. Great interest is aroused by the spontaneous total regression of vaginal (Langhans-Dunger) and other metastases, which had arisen from a typical chorion epithelioma of the uterus. Such regressions have been described by Marchand, Everke and others, after complete removal of the chorion epithelioma of the uterus; disappearance of lung metastases has been described by Risel; in this category also belong the cases of Fleischmann and Hormann in which there was the typical clinical picture of a malignant uterine tumor with metastases in the vagina, and in the second case, also in the lungs; after removal there was regression of the metastases; see also the cases of Labhardt; Michel. v. Velits believed that extensive necrobiosis in these clinically benign chorion epitheliomas was responsible for the regression, but Hormann immediately retorted that the microscopic picture of necrobiosis is part of the characteristic picture of any chorion epithelioma. The biological conditions, and not the histological pictures are therefore important in the true character of this tumor (see also Seitz).

For the microscopic diagnosis in vivo, it is necessary to remember the above mentioned physiological cell and villous wandering, the placental polyps, but particularly the hydatidiform moles. To conclude that a tumor is a malignant chorion epithelioma, it is necessary to view the conditions as a whole, *i.e.*, there must be found plain syncytial and Langhans cell elements (typical form), or perhaps only the former (atypical form), in enclosed masses in the curettings; and furthermore, these masses dare not form definite villi, must be the dominant part of the section and show as compact tumor particles (see also Krukenberg). The diagnosis is made with more certainty in the malignant mole as well as in chorion epithelioma when there is extensive connected cellular overgrowth, not as

in every pregnancy in the form of isolated cells, or, as in retention of the placenta, in masses of chorionic cells, (Kworostansky, Jockers, see also pp. 1716 and 1737), but as an infiltration into destroyed musculature or into vessels which must be filled with polymorphous cells. (See the views of Schickele.) The demonstration of necrosis, hemorrhage, thrombosis and marked leucocytic infiltration helps in the diagnosis of malignancy (R. Meyer). Isolated sections of the covering of villi showing vigorous cell growth do not determine malignancy. This is illustrated very clearly in figs. 866, and 867 in which the clinical course was that of a perfectly benign hydatidiform mole. Durante and Paquy believe there is a differential diagnostic criterion in the condition of the deciduae; if the villi grow excessively, the deciduae are said to be more and more thickened by a defensive small cell infiltration. The more marked this reaction, the more favorable is the prognosis.

The clinical course must be taken into consideration, because, as Aschoff emphasizes, even though the histological examination reveals nothing malignant, the possibility of a malignant chorion epithelioma is not entirely excluded (see also Polano, v. Franque and Hormann, Risel, Veit, lit., Hitschmann and Christofoletti, Schwarzer). The greatest difficulty, however, occurs, because, even when the histological picture is that of a typical malignant chorion epithelioma, it does not necessarily mean that the clinical course of the disease will be typically malignant (see above—spontaneous regression). No certain prognosis can therefore be made. On the other hand, when the rare atypical chorion epithelioma is encountered, it may be possible to give a poor clinical prognosis. In malignant hydatidiform mole, and even in ordinary moles, Neumann believes that the finding of syncytial elements in the chorionic stroma is a criterion of the malignancy of the tumor. But this idea has not been confirmed, and these changes in the stroma may, or may not, be found in malignant moles (Marchand) and, on the other hand, may sometimes be found in the benign type (Pick and others).

Chorion epithelioma-like and hydatidiform mole-like structures in teratomas of the testes (Schlagenhaufer, Wlassow, Steinhaus and others) were discussed on p. 1507, and those in the ovary (Pick) were discussed on p. 1588. To recapitulate, when a chorion epithelioma-like growth is found in a teratoma made up of all three germinal layers, it is merely an expression of the fact that the ectodermal components of the teratoma took on this particular form of epithelial growth. The hydatidiform mole-like structures in teratomas were discussed on p. 1588. These structures, in spite of the fact that they arise from fetal ectoderm, have nothing to do with the malignant chorion epitheliomas of women; the latter are independent tumor formations coming from chorionic epithelial cells, and are

only similar, or somewhat like, the former. As Pick says, the chorion epithelioma and hydatidiform mole of the woman, are in relationship to the carrier as a descendant; the chorion epitheliomas in teratomas are consanguineous. But it might be mentioned that Schlagenhaufer after finding chorion epithelioma-like structures in the testes, drew the conclusion that all chorion epitheliomas are embryonal teratoid products, because isolated blastomeres of the embryo in pregnancy may be a point of origin of a malignant chorion epithelioma. But as Steinhaus remarks, the development of a chorion epithelioma from the epithelium of the villi is indubitably demonstrated, as well as the circumstance that the development of the tumor does not take place into the fetal but into the maternal organism, which is a significant point of difference.

The justification for comparing the similar appearance of the chorion epitheliomatous structure in teratomas with those in the woman is recognized by many; there might be mentioned Risel, Steinert, Emanuel, v. Hansemann, H. Askanazy and others. There have occurred, however, a small number of cases in which isolated, purely typical, chorion epitheliomatous growths, have developed in germinal glandular tumors, in the testes (see p. 1507) as well as in the ovaries (lit. by Risel, E. 11. 1907), and furthermore there are isolated cases in which there was development of a typical chorion epithelioma, obviously from teratomatous anlage, without participation of the germinal glands in other situations in the body, such as in the brain, etc., (Bostroem, Hahn), bladder, (Djewitzki), pineal body, (Askanazy). Pick gave the name of "solid epithelioma" and "solid cystic cystepithelioma," "chorion ectodermale," to a tumor of malignant character which arises in the testes and ovaries and which can be differentiated from the well known forms of typical or atypical chorion epithelioma by the fact that there is an entirely one-sided growth of atypical Langhans cells developing in the teratoma from congenital teratomatous anlage. But objections have been raised from all sides against this interpretation, particularly because the tumor cells do not show the characteristics of chorionic ectodermic cells (v. Hansemann, Michel, R. Meyer, Risel, lit., Chuvin and others). On the basis of his own experience in tumors of the testes, the author shares these doubts and believes they are carcinomas. See under carcinoma of the testes.

Sternberg tried to show that those hydatidiform moles and chorion epithelioma-like structures, which are more or less, and occasionally predominantly, of a teratomatous nature, were perithelial sarcomas (hemangioendotheliomas), in which the peculiar giant cells and syncytial tissue arose from proliferation of the endothelium of the vessels, or from anlage of vessels. Nakayama also described a retroperitoneal tumor in a 38 year old man which was quite identical in structure to the typical

form of a chorion epithelioma, as a blood vessel endothelioma or so-called pseudo-chorion epithelioma. But with a typical chorion epithelioma under the microscope, it is quite difficult to seriously consider this genesis, even though Monckeberg accepted this origin for a number of cases. But Sternberg's work has this particular advantage; all tumors with syncytial and giant cell structure and hemorrhages are not taken to be chorion epitheliomas, since these findings, as the author can confirm, occur also in undoubted carcinomas and sarcomas and in their metastases (see also the cases of Risel, Venulet, and Krompecher). The opinion of H. Albrecht may also be mentioned, which states that the chorion epithelioma is closely related to sarco-carcinomas. For other interpretations concerning the chorion epitheliomatous structure, see p. 1509.

The Puerperal Infectious Diseases

The female genital organs are subject to spontaneous or accidental trauma during every childbirth, and the danger of infectious disease is always present (puerperal fever or wound fever). Tears in the os uteri, the perineum or the vagina, but particularly the large open surface in the puerperal uterine interior, are good portals of entry for infectious organisms.

The invasion and the multiplication of the bacteria are favored by the swelling and moisture of the tissues during pregnancy.

At autopsy, it is important to remember that some severe puerperal infections occur without much participation by the uterus. The infectious organisms wander through the uterine wall and reach the general circulation in a very short time.

The various routes of infection during childbirth or before this process, as well as in the puerperium, are somewhat as follows:

1. Endogenous organisms (self-contained organisms, Hamm), may ascend from the lower parts of the genital canal where they are always present under physiological conditions, into the upper part of the genital passages (from self infection, or better, endogenous spontaneous infection); compare A. Sternberg. Protracted labor, retention of the placenta, retained membranes, tears at childbirth, favor the ascent. But endogenous organisms may be carried from lower to higher portions on hands or instruments (artificial infection with endogenous organisms); see Pankow.

2. Exogenous infectious microorganisms (foreign organisms, Hamm) may be introduced by unclean hands, perhaps those of the pregnant woman herself (autoinfection), or by dirty instruments (artificial infection with exogenous organisms).

3. Secondary infection of the gravid, or more usually, the puerperal uterus, may come from the peritoneum or by way of the blood stream in

pneumonia, angina and other such conditions (Schipkow, Schmitt). The second method of infection is the more frequent.

While the processes which are ordinarily called "infection," consist of the invasion of bacteria into the living tissues, there are other processes, and these not rare, in which decomposition occurs under the influence of bacteria in partly separated and undernourished tissues (*e.g.*, retained secundines infected from the vagina) and poisonous substances are absorbed into the venous circulation, whereas the bacteria themselves do not show invasive characters. This latter process is called putrid intoxication (sapremia), or "retention fever." The saprophytes, particularly of the proteus variety, but also bacillus pyocyaneus, micrococcus tetragenus and others, are the most important of these organisms. The possibility of pure cases of sapremia is undoubted (see also Burckhardt); with removal of the decomposed poisonous masses or retained lochia (lochiometra) by currettage or douching, the progress of the intoxication is stopped and the fever disappears. Very often, however, this is followed by infection of the blood stream, *i.e.*, the passage of bacteria into the blood (bacteremia), an occurrence which on the whole does not have grave consequences and may be accompanied only by a chill, when a considerable number reach the blood stream and their endotoxins are freed by bacteriolysins (toxemia, septicemia). If the blood infection is not the most important part of the picture, these cases of sapremia are due to the local production of poisons. Recently there has been much controversy concerning the sharp separation of putrid intoxication and septic infection, *i.e.*, sapremic and septic fever (Schottmuller, Hamm, Bondy and others), but many authors do not like to discard the idea of resorption fever and pure toxemia (Koch, Friedrich, Fromme, and others). In septic infection, the bacteria themselves excrete toxins which, on the one hand, form the most prominent lesion at the situation where the bacteria are established, and, on the other hand, produce the clinical picture of sepsis from the absorption of toxins coming from the portal of entry (toxemia), and from the bacteria in the blood stream itself (bacteremia). It is, therefore, a general infection of the organism which is manifested, among other things, by headaches, fever, disturbances of the central nervous system, heart failure, and so on, and is anatomically disclosed by severe damage to many parenchymatous organs, swelling of the spleen and other similar conditions. If metastatic or embolic processes (abscesses, infarcts) are found in the various organs, the condition is spoken of as pyemia, or since there is always a septicemia, as a septico-pyemia.

Instead of pyemia, Lenhartz proposed the name "sepsis with metastases." Schottmuller reserved the term sepsis for the following circumstances: when there is a "septic focus" (not *eo ipso* the portal

of entry) in the body, *e.g.*, an endocarditis, or cholecystitis, or an infected uterus in atony or infected abortion, from which bacteria are constantly or periodically liberated into the blood stream so that this invasion is felt subjectively and is shown objectively. There may also occur metastases (abscesses, septic and bland infarction); in this case the term "pyemia" may be used or "sepsis with metastases." To characterize the findings on the autopsy table, however, the names "sepsis" and "pyemia" cannot be spared. See also Nacke concerning sepsis and pyemia in their clinical aspects.

The streptococcus pyogenes is the chief organism concerned; it is found in the most severe as well as in the most mild puerperal infections, undoubtedly because of the wide differences in virulence which these organisms assume. According to Schottmuller, Bordet, W. H. Schultze, Fromme, Henkel and others, there are three chief groups of human pathogenic streptococci; streptococcus longus seu erysipelatos, forming punctate white colonies with bright rings of hemolysis on blood agar plates; streptococcus mitior s. viridans, small green colonies without visible hemolysis on blood agar; and streptococcus mucosus, mucoid colonies without hemolysis on blood agar. Fromme attempted to use the degree of hemolysis as an indicator of the pathogenicity of the streptococcus (see also E. Sachs), but others feel that the degree of hemolysis cannot be thus correlated (Polano, Zangemeister, lit., Burgers Lamers, Traugott, lit., Goldstrom). Less commonly there are found: staphylococcus aureus and albus; colon bacilli in combination with streptococci, the most frequent mixed invaders; pneumococcus (see Bondy), tetragenous; various kinds of anerobic bacilli, among them the frequent inhabitant of the vagina,—the streptococcus putridus, (Schottmuller),—as well as specific organisms such as the gonococcus (from a previous cervical gonorrhea), the diphtheria bacillus (Bumm), tetanus bacillus (Rubeska, Spiegel, lit.), and the influenza bacillus (very rare, see Thaler and Zuckermann). It is of great practical importance that gonococci which may have been resident but latent in some parts of the genital canal for long periods of time, or which have produced a "residual gonorrhea" (Sanger), may bring on acute complications during childbirth or, particularly, late in the puerperium (Leopold, Fehling, Fritsch, Bumm, Bischoff and others), and also that the husband may be freshly infected (Lowenheim). The bacteria ordinarily present in a healthy genital canal cause no trouble in an uncomplicated labor. In pathological labor, however, they may cause not only septic decomposition processes, but also take on infectious characteristics (autogenous puerperal self infection, see Zangemeister); a disturbance of the equilibrium between the immunity of the body and the virulence of the microbe is also spoken of, in which the latent vaginal and cervical

organisms are suddenly allowed to take on increased virulence, or to manifest their native virulence (Salomon). The acid vaginal secretion (see p. 1691; concerning its chemistry during pregnancy, see Zweifel), is made alkaline by the alkaline amniotic fluid (Bengelsdorff), blood and the lochia; its "bactericidal properties" are destroyed, and thus the development of pathogenic organisms in the vagina is favored. On the other hand, Bumm and Sigwart found streptococci in the vaginal secretion in 74% of pregnant women, and in 75% they were recovered in the lochiae from the vagina; pyogenic staphylococci were found by Amersbach in the vaginal secretion of 48% of pregnant women, in which case, these "bactericidal properties" of the "vaginal secretion" cannot be given too much credit. Even on the third day, the lochiae of a healthy woman are infectious. The cavity of the uterus remains free of bacteria for a longer time in the normal puerperium (Doederlein, Winternitz), at least until the beginning of the first week; at later times, usually, even with no fever, it was found to contain organisms (Burckhardt, Wormser), streptococci in many cases (Schenk and Scheib and literature by von Herff). According to Loeser, however, bacteria enter the internal surface of the uterus in 25% of the cases two days post partum, and it is not the quality of the organisms, but the reaction of the tissues and their resistance which determine a febrile or afebrile puerperium.

As portals of entry, we find pathological changes local or diffuse; the infectious organisms reach the lymphatic spaces or even larger lymph vessels or veins and thus ascend further and further into the body of the uterus. The portal of entry, however, may be entirely free of local changes, and in spite of its apparently healthy appearance, allow organisms to enter the blood stream and produce a general infection. Puerperal ulcers are usually infected by moulds, and are yellow gray wounds on the vulva, vagina, or the os. They are usually merely surface processes, in which saprophytes are present in dead tissue; the latter is expelled after a superficial reactive suppuration. But if infection takes place with pathogenic organisms the ulcer takes on diphtheritic and gangrenous characters and a phlegmon of the neighborhood or a generalized infection may follow.

Infections of the uterus involve the mucosa or the muscularis or both. The peritoneal covering may also take part.

Puerperal Endometritis.—In order to understand this condition it must be remembered that the inner surface of the normal puerperal uterus (see p. 1717) is lined by a villous, tightly adherent layer of deciduae, which is separated in tags during the puerperium. A few days post partum there are still localized remnants of loose, fatty degenerated, and necrotic deciduae of yellow gray or pale gray color. The beginner may

mistake these for inflammatory pseudo-membranes, but they are easily washed off with water. When organisms of decomposition ascend in the normally necrotic parts of deciduae, especially if the latter hang from out the os into the vagina, the ensuing decomposition produces an evil smelling mass which can easily be scraped off; the lochiae themselves are evil smelling. The organisms are usually mixed anerobic and aerobic, saprophytic and pathogenic (see Sackenreiter), and do not enter the depths of the tissues but remain on the surface and are separated from the deeper layers by leucocyte—containing granulation tissue growth from below. According to Schottlander, however, a few bacteria always enter the blood stream. This mild puerperal infection is called "localized putrid endometritis" (1). Pathogenic organisms which enter by spontaneous ascension (see above) as well as by intrauterine manipulation with the fingers or instruments, naturally also may remain localized in the surface layers; it is, however, more typical for them to invade the tissues. The bacteria of decomposition and pathogenic microorganisms may be combined. The invasion of pyogenic streptococci gives rise to septic endometritis (2), with suppurative or fibrinous exudation, diphtheritic membrane formation and gangrenous destruction of the mucous membrane (diphtheritic endometritis). Opening the softened uterus of severe cases, reveals a bloody ichorous fluid in the cavity and the whole endometrium changed to a thick, green gray or green brown discolored mass, which, as shown on cross section, is partly detached and partly adherent to the endometrium. Scraping with the knife usually does not allow of separation to a smooth muscularis. The masses consist of necrotic tissue, fibrin, leucocytes and organisms. Occasionally these changes take place only at isolated points, such as the situation of the placenta or in tears of the cervix and their immediate neighborhood. If the endometrium has been destroyed to a foul smelling yellow brown or yellow pulp which can usually be scraped off with the knife, the diagnosis of deciduae may be quite difficult (microscopically: large decidual cells!). Further invasion of the organisms to the lymph and blood vessels leads to diffuse peritonitis or diffuse parametritis or to a general septic or pyemic infection.

Puerperal inflammations of the uterine wall may be localized principally (1) in the blood vessels, (2) the lymph vessels, or (3) the parenchyma.

(1) **Puerperal metrophlebitis** is caused by invasion of infectious organisms, usually streptococci, mixed perhaps with decomposition organisms, into the veins, most frequently from the previous situation of the placenta. The first infection is often in (aseptic) thrombi, present in the vessels for a long time because of insufficient contractions of the uterus; instead of the normal thrombi (usually coagulation thrombi), there are masses totally or partially softened, ichorous and dirty brown

in color. The walls of the veins are invaded and appear yellow and thickened. Secondarily, the phlebitis leads to thrombosis in other situations; most of the thrombi are very soft and surrounded by pus. Infected thrombi then spread into the peripheral veins of the uterus, which are usually empty or filled with fluid blood.

But, on the other hand, the microorganisms may enter the walls of the veins directly, produce a phlebitis from the inside or from the outside of the vein with secondary thrombosis. The thrombophlebitis then spreads through the broad ligaments into the plexuses of the ovarian veins and from these into the inferior cava or into the left renal vein. Other routes are: through the pampiniform plexus into the hypogastric vein, to the common iliac, from here by retrograde transport into the crural veins (phlegmasia alba dolens); Olshausen and Fehling believe this is a phlegmon coming from the parametrium and spreading into the legs, but Kroemer believes it follows a retrograde stasis of lymph from a "perivenous reaction" in a phlebitis of the veins of the pelvis, a process which almost always leads to an obstructive thrombosis of the veins of the pelvis and the legs. From the pampiniform plexus the branches in the tubes and the ovaries may be thrombosed. The separation of larger or, more usually, smaller clumps leads to embolism through the inferior cava to the right heart and the lungs; furthermore the very smallest clumps of bacteria may pass through the veins and capillaries of the lungs and enter the greater circulation. Abscesses from the presence of both pus and decomposition organisms lead to ichorous suppuration everywhere (puerperal pyemia). Surgical ligation of veins to prevent further passage of emboli or thrombi has yielded good results in some cases (Martens, lit.). This condition is also spoken of as "venous sepsis" or as the "thrombophlebitic form of puerperal fever." On rare occasions, it may come from the vagina. The author has also seen cases following probable artificial abortion in which the phlebitis involved particularly the cervix, the walls of which were of the thickness of a thumb and had become spongy from the infiltration of ichorous material in the veins; ichorous phlebitis phlegmons of the parametrium are followed by peritonitis.

(2) **Puerperal metrolymphangitis** occurs coincident to thrombophlebitis or as an independent entity. The process involves especially the subserous lymph vessels in the neighborhood of the cornuae of the uterus; the yellow walls are infiltrated by pus, and the lumena are filled with yellow puriform material containing extraordinarily large numbers of micrococci. The tissues surrounding the lymph vessels, *i.e.*, the myometrium, are often phlegmonous. The content is pus or a leucocyte-rich coagulated lymph. Since the sagittal section through the anterior wall, as ordinarily made of the uterus at autopsy, may not show these

changes, it is recommended that many flat sections be made, particularly through the subserous layers. The walls are not usually ulcerated except in chronic cases in which the lymph vessels become abscess cavities (uterine abscess, lit. v. Franque and Suave, Pielsticker, lit.). The lymphangitis spreads to the adnexa, the lymph vessels in the broad ligament appear injected, or like strings of pearls (see p. 174), and become as thick as lead pencils; abscesses in this neighborhood are readily understood, since the lymphangitis is always accompanied, even though in varying degree, by a phlegmon of the tissues through which the lymph vessels pass. A fibrino-purulent or suppurative peritonitis practically always follows, from which a pleuritis and a pericarditis may be developed. The phlegmon may extend in the retroperitoneal connective tissue even as high as the diaphragm. This is the "lymphangitic form of puerperal fever." The point of origin, in addition to the cavity of the uterus, may be in infected tears of the external genitalia, the vagina and os, the lymph vessels of which communicate with those in the walls of the uterus, with those of the peritoneum, the tubes and ovaries; but the infection may also occur without demonstrable injury, as (1) *e.g.*, from the pregnant woman herself.

(3) A **phlegmonous metritis** takes the form of a more or less extensive softened yellow or gray red edematous infiltration, or death of the tissues by bacon-rind-like necrosis, or suppurative infiltration and the formation of abscesses. The routes by which the phlegmon is spread and by which it is established are the spaces in the tissues. There are also forms in which the involvement of the larger lymph vessels is dominant (as in *b*), and others in which diffuse spread in the tissue spaces and meshes prevails (as in *c*), or both forms may be combined. Phlegmonous metritis may, among other conditions, be initiated by gonorrheal infection in the puerperium. By dissecting metritis is understood a condition in which larger or smaller masses of necrotic tissue are separated from the walls and are expelled (lit. by Beckmann and v. Franque).

Co-involvement of the Parametrium.— Deep infected cervical tears, deep perineal tears, pressure necroses of the neck or the vaginal tissues, frequently lead to an infection of the parametrium, in which the inflammation is often localized (see under parametritis, p. 1645), but which in severe cases may become a purulent or ichorous affair with perforation into the neighboring organs A fatal result is uncommon. In contradistinction, the rare diffuse parametritis is found only in the most severe infections of the endometrium or the vagina and is a part of the picture of phlegmonous puerperal infection which may arise even in pregnancy or immediately at childbirth. Extraordinarily virulent bacteria such as streptococci, colon bacilli, the bacillus of malignant edema and others

producing gas, are the exciting organisms; among the latter is that known as the "bacillus phlegmones emphysematosae" (Eug. Fraenkel, see also Heyde, W. H. Schultze, Ghon and Sachs and von Hibler, Hanser and Coenen and particularly Bingold, lit.), which is held by many to be identical with the bacillus aerogenes capsulatus of Welch-Nuttal (Goebel, Doblin). These bacteria are found as etiological factors in the so-called tympania uteri (physometra). The colon bacillus has also been held responsible (Gebhard), justly or unjustly. At least in tympania uteri, gas forming aerobic bacilli are found, which are culturally similar to the colon bacillus, but nevertheless have certain points of difference (Lanier). A phlegmonous or gangrenous inflammation spreads from the endometrium into the uterine wall; the latter is swollen, edematous, doughy, particularly in the region of the blood vessels; it is saturated with gas forming bacilli which give rise to enormous gas bubbles crepitating on pressure. From here, or from the vagina, all the tissues of the pelvis are infected and appear puffy, bloody, and contain similar gas bubbles. Following connective tissue layers, bacteria spread, in one case, into the pelvic musculature, and in the other case, into the nates, the perineum, and along the crural vessels into the thigh. This is the so-called gas phlegmon or gas gangrene in which all the tissues become doughy and crepitate from the enormous number of gas bubbles. In an upward direction, the infection spreads through the retroperitoneal tissues by continuity through the diaphragm into the pleura, the interstitial tissue of the lungs, mediastinum and the pericardium; to which there is added dissemination through the blood and lymph vessels, so that finally bacterial emboli can be found in all the organs, even in the retinal and chorioid vessels. If the patient does not die previously from toxemia, abscesses are developed, or when gas forming organisms are present, innumerable small gas bubbles, so that all the organs are punctured by holes like a sponge. So much gas may be present in the blood itself that differentiation from air embolism is quite difficult. Even the skin of the fetus may be crepitant and emphysematous (Doblin). The most severe degrees of diffuse parenchymatous degeneration in all the organs are constant findings. Peritonitis is practically always present. The patients die, usually in a few days, from the severest of intoxications. In milder cases, the infection may possibly localize, as reported in a few instances.

Involvement of the Peritoneum.—A diffuse peritonitis is the most frequent manifestation of grave puerperal infection, usually from a septic lymphangitis coming from the vagina, the os, or the uterus itself. But in other cases the streptococci produce localized necroses of the wall of the uterus from which the peritonitis originates. In still others, the organisms reach the peritoneal cavity from an undiscoverable portal of entry and

produce a quickly fatal ichorous peritonitis with very little exudate. In rapidly fatal cases, the peritoneum is merely red and cloudy and only in the neighborhood of the pelvis; in cases of longer duration the atonic intestines are dilated and covered with fibrin and pus (see p. 863).

On p. 1748 we have mentioned the possibility of secondary hematogenous infection in the puerperal uterus. From other points of origin, such as a pneumonia, angina, there may occur a lymphogenous or a hematogenous purulent peritonitis with an intact puerperal uterus; such cases of non-puerperal sepsis in the puerperium may be quite difficult of judgment at autopsy (see Schmitt); they may be called pseudopuerperal fever.

For the pathological anatomical aspect of this puerperal process, see also Halban and Kohler, Wien, Braunmuller, 1920.

The Breast

Embryology. Anatomy.—The first traces of breast anlage were discovered by O. Schultze in fetuses at the end of the first month in the form of a milk-line or a milk-stripe, on both sides of the mid-line between the upper and the lower extremities; the stripes are of higher epithelium, which multiplies and becomes the milk glands. The actual breast anlage are therefore epithelial thickenings arising from a local increase of cylindrical cells (rete cells) in the embryonal epidermis. These form prominences which grow into the depths, and are later transformed into the areola and the nipple (papilla, mammilla). From the germinal layer of the lowermost epithelial cells of this "gland area," secondary growths produce isolated, at first solid, ducts which later become hollow and branched; their blind ends are the so-called gland vesicles; at the same time the connective tissue grows apace, (literature concerning embryology by Teigeler, Bauer). The gland vesicles and ducts are composed, just as the sweat glands (sweat gland nature of the breast, see Pinkus—and Moll's glands of the eye lids), of two cell layers, the inner of cylindrical epithelium, the outer of contractile cells which are cuboid in the ducts and lower in the vesicles (basket cell layer). Kuru, Benda, believe these basket cells are smooth muscle, also that they originate from capillaries; Letulle describes them as "subatrophic;" La Roy believes they are endothelium belonging to the membrana propria; (for more details, see Chalatow). The connective tissue membrana propria lies on the outer surface of the basket cell layer, but closely adherent to it; on the outside of this the adventitia is composed of tender connective tissue, rich in leucocytes, plasma cells and vessels, and is metachromatic during the developmental period of the glands (see p. 1772), but not in the completed breast (see Kuru). The tough, poorly cellular intercanalicular or interstitial connective tissue lies

between the gland acini as a soft yielding pillow. Between the isolated gland complexes, is the fat. In the newborn, the breast is composed of about twenty ducts. The larger ones are lined by cylindrical or layers of squamous epithelium, and contain a milk-like fluid composed of desquamated cells and decomposition products, the so-called witch's milk, a substance which, however, has nothing whatever to do with true secretion (Raubitschek). But the ducts may be considerably dilated.

In the male, the gland reaches its maximum size at about the twentieth year, but remains a very circumscribed structure. About the fiftieth

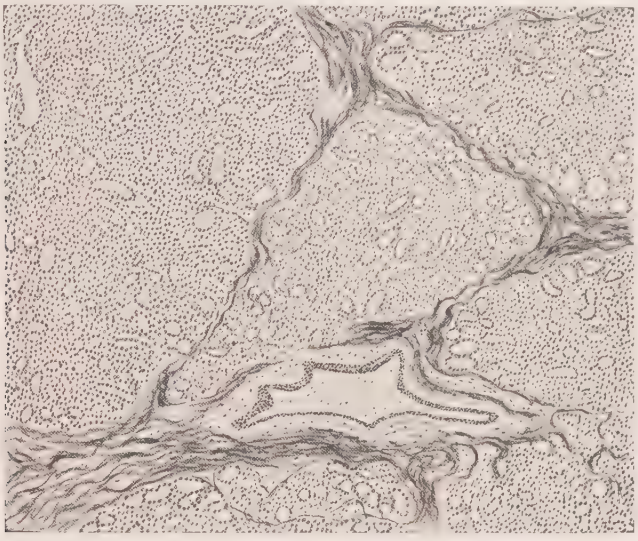


FIG. 878.—Lactating breast from woman 28 years old; coarse interstitial connective tissue divides the gland into lobules, and surrounds a duct; gland vesicles and ducts contain secretion. (Med. magn.)

year, there are only isolated, occasionally ectatic, ducts surrounded by much hyalin connective tissue giving the section surface a roughly nodular appearance.

In the female, the resting gland is composed chiefly of broad fibers of poorly cellular connective tissue in which isolated gray red gland masses are scattered. An individually different growth of glands takes place at the time of pregnancy and reaches its maximum at the time of lactation. New side branches are formed from which arise other side branches connected with acini; the cells become higher, cylindrical, and thicker, and the basket cells are lacking; in their place are capillaries. When the production of glands is marked, the section surface is nodular, pale gray red and similar to the section surface of a salivary gland. The soft, moist connective tissue is also considerably increased but is thickly studded with

glandular tissue; the rough, interstitial, slowly growing connective tissue then appears in the background, but occasionally sends prolongations around the glandular complexes. Secretion can be demonstrated by pressure, even in the second month of pregnancy; just before lactation, and, of course, after this is established, pressure upon the glands yields a thick, creamy yellow fluid, while the section surface at the height of lactation is bluish white and liberates relatively transparent milk. In rare cases, in the newborn (a hormone coming from the mother is said to be implicated), but also in adults, outside of pregnancy, thus at the time of puberty, (even in males), a small amount of secretion can be demonstrated; this also is spoken of as witch's milk. The milk is secreted by the epithelium of the glands which, as a rule, remains intact. According to the observations of Arnold (lit.) concerning the processes of secretion, tiny fat granules are found in the breasts of pregnant and lactating women, at certain places in the basal portion of the cells and in the neighborhood of the nuclei. Since they occur at definite structural places of the cell, these granules are believed to be the result of a synthetic process. They grow into secretion droplets composed mostly of fat or albumen or both, and produce small holes in the protoplasm, the so-called secretion vacuoles, which can be demonstrated after extraction of the fat as well as after expression of the secretion into the lumena of the glands. The nuclei do not take part in the secretory process. The fat secreted by the breast is a mixture of that of the food, or directly from it, or from the fat depots; it is brought to the breast in soluble form, and does not appear intracellularly via fatty degeneration of the protoplasm, as Virchow assumed. The colostrum secreted in the first two or three days after childbirth, and in small amounts even in the second half of pregnancy (see, among others, Metzner), is a mixture of watery, translucent or creamy opaque secretion, containing microscopic fat droplets of varying sizes, isolated fat-containing epithelial cells, leucocytes and the mulberry-like colostrum bodies with nucleated cells which are filled with yellow stained or unstained fat granules; these are also called fat droplets. Ordinary fat droplet-cells with a flat contour and recognizable nucleus also occur. Formerly, and even today by some (de Lange), the colostrum cells are believed to come from epithelium, but A. Czerny showed that they are mostly leucocytes which wander into the gland spaces from the blood stream as soon as milk is produced. They are not emptied toward the exterior and therefore are not peculiar to this milk; the leucocytes found in large numbers around the acini and in the interacinar and interstitial connective tissue take up the secreted fat by way of phagocytosis and by synthesis (Arnold), divide it, and take some of it by way of the lymph stream to the lymph nodes. Women who do not nurse have many colostrum bodies after a few days. According to Berka

most of the colostrum cells are large lymphocytes coming from the connective tissue stroma as infiltrates; Thomas, (lit.), differentiates polymorphonuclear and perhaps histiogenic macrophagic mononuclear colostrum cells. The destroyed gland cells are taken away by phagocytosis. A retention mastitis readily occurs, (see p. 1761). Milk is microscopically an emulsion of fat droplets of fairly even size (2–5 microns), held in a clear fluid. Isolated leucocytes and colostrum bodies are also found inconstantly (Bab). Chemically, the milk consists of 88.9% water, the solid substances of fat, casein, milk sugar and salts. Colostrum contains more serum albumin than casein and is richer in solids than milk.

For the physiology of lactation, see Pfaundler in Sommerfeld's Handb. d. Milchkunde, Wiesbaden, 1909.

Montgomery's glands (areolar glands), under the skin of the areola, are not sebaceous glands (Waldeyer-Jossel), but accessory milk glands, the excretory ducts of which empty in the neighborhood of the nipple. According to others, breast lobules and ducts might be combined with sweat and sebaceous glands. They enlarge during pregnancy and in lactation. In the skin of the nipple are a number of microscopic sebaceous glands, which empty in the depressions between the papilla of the prominences next to the ends of the milk ducts.

The Relation of the Breasts to the Other Sex Organs.—The strongest impulse to grow is received by the breast during pregnancy, but it is debated whether the source of the hormone stimulus is the placenta (Halban), the corpus luteum or the entire ovum (Aschner, lit. by Herrmann). Occasionally a lactation hypertrophy in young girls is seen coincident with carcinoma of the ovaries (see Saenger, Askanazy, Schmincke); the thought is expressed that material in the ovarian tumor acts like a hormone. See also Ebeler concerning pathological breast secretions in various gynecological diseases. See Scherbak concerning the internal secretion of the breast. After castration, or when the ovarian function is destroyed, atrophy of the glandular substance takes place; this may be hidden externally by the overgrowth of fat.

Malformations.—There may be mentioned: congenital absence of one breast, amastia, (very rare); congenital diminution in size of the areola or the entire gland (micromastia). Gynecomastia is a marked, usually bilateral, painful development of the breasts in males, a macromastia, of course, made up only of ducts with blind ends and without acini. There is said to be an hereditary tendency; other genital malformations are frequently coincident. (Lit. by Burgi, Schon, Schneller.) There also occur supernumerary nipples, (hyper- or polythelia); according to Iwai, as many as six have been observed; according to Jellinik, they are more frequent on the left side; supernumerary breasts (polymastia or hyper-

mastia, accessory breasts) also occur; the former are situated either on the breast itself, or receive their ducts from supernumerary breast glands. Aberrant breasts are met in many situations; they have even secreted milk; they may be situated above or to the outside of the ordinary breast, as well as in the axilla (lit. by Kayser, Hug, Seitz), in which they may be mistaken for lymph nodes, and also, according to Seitz, for hypersecreting skin glands (sebaceous and sweat glands) during the puerperium; or they

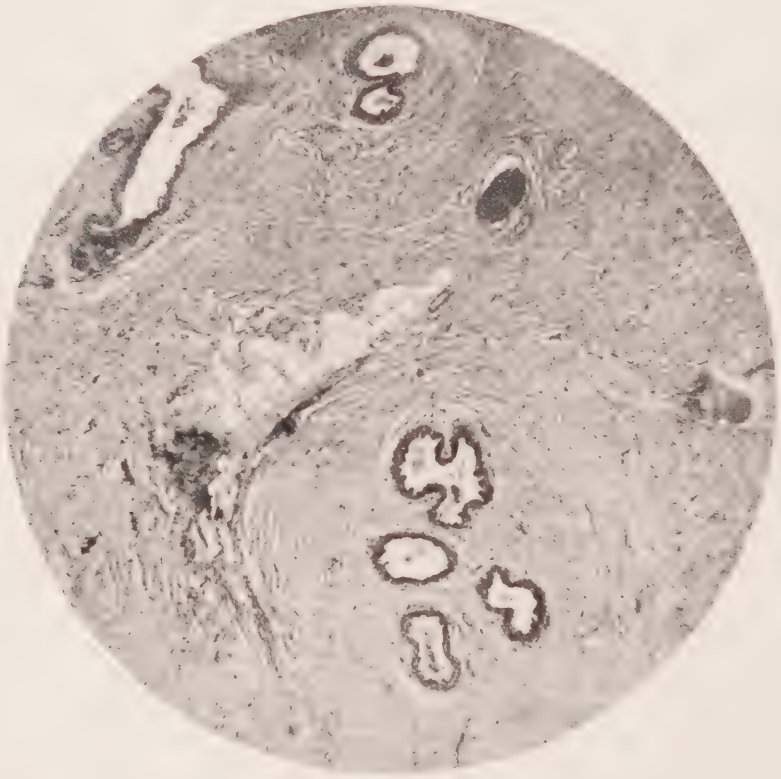


FIG. 879.—Gynecomastia. Section from an enlarged breast of a man 22 years old. Present and growing for about two years. Sections revealed structure as above, namely, ducts surrounded by a periductal connective tissue and fat. There is no evidence of the formation of acini nor was there in any of the other specimens examined. Gynecomastia, therefore, is an increase in size, and probably also in number, of the ducts with accompanying increase in the connective tissue. × 60. L. H. #17335.

may be below and inside of the normal breast, or distant from this structure, on the abdominal walls, on the thighs, in the labium majora (observation by the author) or on the back. The author saw a breast of considerable size in the inguinal region to the right of the mid-line. They occur in men as well as women (lit. by Schneller) and are often found among sisters. An atavistic tendency has been invoked as a cause

(Leichtenstern, lit. and others). (Adenomas or fibroadenomas, also carcinomas, may arise in these misplaced breasts.)

Circulatory Disturbances and Inflammations.—Congestive hyperemia occurs during menstruation and in the beginning of lactation. The breasts swell, become warm and red. Hemorrhages occur from trauma. The blood may become encapsulated, and after absorption of the extravasation may leave a cyst without epithelial lining, but with pigmentation on the inner surface. But hemorrhages also occur in true cysts already present and lined by epithelium. In various tumors, particularly sarcomas, as well as in inflammation, hemorrhages are prone to occur. If the blood breaks into an excretory duct, it may be emptied in part through the nipple (bleeding nipple); this occurs in cystic mastitis; a 35 year old woman bled four months from the nipple, and a 50 year old woman bled from the nipple for four years, (author). (Lit. by Moraller, Weishaupt, L. Fischer.)

Gruber describes hemorrhagic infarction following stasis and thrombosis in the veins.

Inflammation of the areola (thelitis), with cracks or fissures and small ulcers, is not uncommon in lactating women from the suckling movements of the child. It may be the starting point of a mastitis. Wallart saw gangrene of the nipple in the puerperium following the administration of orthoform.

Inflammation, Mastitis.—There may be differentiated: (*a*) simple parenchymatous mastitis, (stasis mastitis); (*b*) infectious mastitis.

(*a*) The mastitis of stasis is due to irritation from retention of secretion, particularly in lactating women. The stasis, which may appear on the first or second day or later if the emptying of the breast is hindered by any cause, may be accompanied by mild increase in body temperature (milk fever). Since stasis favors infection, there is danger of secondary suppuration from this simple mastitis. Stasis of milk alone does not produce this condition.

In the same category, belong the cases of mastitis in the newborn, the pubescent mastitis of both sexes, and the stasis in many women at menstruation. In these cases also, stasis favors the entrance of infecting organisms. In the mastitis of the newborn, infection possibly comes from the maternal vagina; in the swelling of the breast in newborns, Gruber found hyperemia and hematopoietic foci. There are observed redness, serous infiltration and painful swelling. These usually regress in a few days; in rare cases, however, abscess formation occurs.

(*b*) In infectious mastitis, pyogenic organisms invade the breast; the portal of entry is usually (1) through the nipple, (2) through the milk ducts, (3) alongside of the milk ducts through the lymph channels, but

in rare cases, a mastitis may occur (4) by metastasis in a pyemic, usually puerperal, process, or in typhoid fever (Madelung, lit.) or even from the thorax (empyema, caries). There are differentiated (Bumm):

1. Infectious parenchymatous mastitis in which, as a rule, the breast is attacked during lactation. The infective organisms are usually staphylococci, which have entered through the nipple from the outside and have passed up through the excretory ducts (galactophoritis); reaching the ends of the ducts, they destroy the epithelium, call forth a migration of leucocytes and may finally even infiltrate the neighborhood. There thus arises a series of lobulated painful nodules as large as walnuts usually in the lower outer quadrant; later the process diffuses and the entire breast grows hard and thick. The inflammation regresses, or progresses to abscesses.

2. Infectious interstitial mastitis or phlegmon of the breast is usually caused by streptococci, which enter from the nipple and spread by way of the lymph channels alongside the ducts into the depths of the interstitial tissue and bring forth cellular infiltration and inflammatory edema. The glandular parts are involved secondarily. The interstitial inflammatory process quickly leads to painful reddening and diffuse swelling; resolution may occur, or tissue suppuration (abscesses) ensues in which the gland acini themselves are partially or totally destroyed.

The abscesses are seldom smooth, but are usually irregular and unevenly burrowed. The pus may contain necrotic bits of tissue.

Abscesses between the skin and the glands are called "antemammary;" those between the muscles and the breast are called "retromammary;" the latter may have other causes, *e.g.*, caries of the ribs (see Hardouin and Marquis); both conditions are sometimes called "paramastitis." The consequences of ascending mastitis are: (*a*) spontaneous perforation through the skin to the exterior with healing by scars, with or without nodular hardenings; (*b*) internal perforation into an excretory duct, when the pus is emptied through the nipple (very rare).

The bacteria present in the secretion of the healthy female breast (usually staphylococcus pyogenes albus) probably enter the ducts from the skin (Honigmann and others). It is questionable whether most pathogenic bacteria can be "excreted" through the healthy breast, but the possibility has been demonstrated for tubercle bacilli, at least, in animals (Hirschberger); in humans, proof is wanting (Fuster). According to Basch and Weleminsky, only those bacteria which can produce hemorrhages or other local conditions in the excretory ducts are excreted in the milk. The question of the passage of toxins, (see Romer), is not answered by these experiments. (*c*) Another end result may be chronic mastitis; repeated perforation to the exterior leaves suppurating fistulae;

if ducts are eroded they become milk fistulae, or the thickened caseous masses of pus remain in the tissues as hard nodules. (*d*) In rare cases, a general sepsis in the puerperium arises from the breast. The author has seen such a case with a rapidly fatal ending (mixed infection by staphylococci and bacillus pyocyaneus); it was particularly remarkable because there were innumerable hemorrhages in the skin, the mucous membranes, the serosae, (in pyocyaneus infections, hemorrhages are frequently observed). (For general infections by bacillus pyocyaneus, see Eug. Fraenkel, V. A. 183, 1906; he especially describes the hemorrhagic vesicles in the characteristic exanthemata of the skin, ekthyma gangrenosum.) Also see Unna-Festschrift Bd. 1, 1910; also Z. f. Hyg. 72, 1912 and 84, 1917 (lit.). (See also Justi, l. c. p. 608.)

In chronic mastitis, the foci of indurated tissue contain small cell infiltrates, and cystic dilated ducts and gland acini (chronic cystic mastitis). The breast is enlarged at first, either partially, and often in pyramidal form, because one particular group of lobules is involved (difficult to distinguish from carcinoma), or diffusely, in which the consistence, however, is not even, but uneven, especially in the neighborhood of cysts containing fluid under high tension; in this case also, they are easily mistaken for carcinoma. The breast, however, partially or entirely, undergoes shrinkage (cirrhosis of the breast). The cysts are usually small, but may be of considerable size, and their contents vary from watery, mucoid, cloudy, cream-like fluid to butter-like caseous, brown or green grumous material. The epithelium of the glands may show, among other things, hyalin or hydropic changes, proliferation and desquamation.

This may lead to obliteration of ducts (Langhans', Samelson-Kliwansky, lit., Ingier, Horz). The material behind the obstruction stagnates, and occasionally contains microscopic granules and droplets of doubly refracting substances and bundles of fatty acid crystals, about which there is an active foreign body giant cell proliferation with many phagocytes which take up lipoid substances (the so-called pseudo-xanthoma cells). Extensive granulation tissue is established, while the epithelium disappears. This condition can easily be mistaken for actinomycosis and tuberculosis! Similar masses occur occasionally in a retention mastitis to which infection has been added.

Under the name of "presenile involution," Hedinger describes those diseases of the breast which are often called chronic mastitis and which are clinically manifested by pain and tenderness (mastodynia) in youthful individuals. Microscopically, however, the changes are the same as in senile involution (Berka).

A chronic mastitis may furnish the background for a carcinoma; on this point all authors seem to be of unanimous opinion, even though the

views concerning the exact position of chronic cystic mastitis are still widely divergent (see p. 1765). Clinically, as stated, it is easily mistaken for carcinoma. Histologically, the differentiation may be difficult, especially when the epithelial proliferation, particularly in the dilated excretory ducts, forms papillary prominences or breaks through acini (see fig. 880), or when they occur on the inner surface of cysts. The question is the fundamental one of distinguishing between hyperplasia and tumor formation. The difficulties are increased when there is added to the picture the change to squamous epithelium, occasionally seen in duct carcinomas (see fig. 894). Irregular distribution of epithelium in the

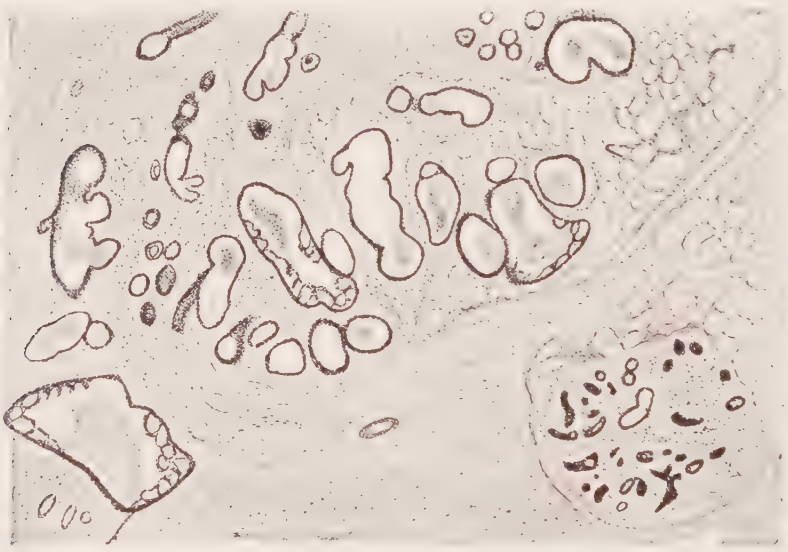


FIG. 880.—So-called chronic cystic mastitis; to the right below, gland lobule with beginning dilatation of lumen and loose cellular periacinar tissue resembling a beginning adenofibroma. Elsewhere, enlarged ducts and acini embedded in cellular connective tissue; here and there fatty tissue, especially to the right. Papillary and broken proliferations of epithelium enter the dilated ducts; in the lumena, detached, rounded pale epithelial cells and homogenous masses. Unmarried woman 50 years old. (× 18.)

excretory ducts must be viewed with suspicion; it awakens ideas of tumor. The clinical and macroscopic diagnosis is often uncertain; the microscopic diagnosis must be made with great care. See also Josselin de Jong concerning the so-called precarcinomatous changes.

Maladie cystique de la mamelle (Reclus) has also been called cystic breast or mamma cystica, and develops frequently, but not always, in both breasts, either coincidently, or first in one and then in the other; innumerable, usually small, rounded cysts from the size of peas to beans, seldom larger, are the characteristic findings. The entire breast, the skin of which is easily movable, is changed into a conglomeration of larger and smaller nodules which feel like a bag of shot or marbles; the breast as a

whole may be considerably enlarged. The author has seen cysts as large as a small fist, along with those the size of walnuts or cherries. The above mentioned name should only be used if the cysts are predominant in the macroscopic picture (just as in cystic kidneys). These cases, however, are not very frequent, although the classical ones have been identified with others considered chronic mastitis with cyst formation, in which the cysts play only a slight and often unimportant part in the enlargement of the breast. The cyst contents are usually greenish brown, viscid or serous; here and there it may be creamy or atheromatous, and in isolated cysts, may be bloody. There are a number of opinions concerning the origin of this process. Konig believes it is a chronic mastitis with the formation of cysts; in general, Borst shares this opinion. We also agree that this idea is correct in many cases (see Lichtenhahn, lit., also Thevenot and Alamartine, Sommer, Kudjii). On the other hand, von Saar believes that the occurrence of a cyst-forming chronic mastitis (Konig) has not been demonstrated and pleads for its neoplastic epithelial nature. Theile (lit.) is also opposed to Konig on the ground that it is a primary epithelial overgrowth. Therefore the condition is also spoken of as "cyst adenoma" or "polycystoma" (Brissaud and others). According to Brissaud and Sicre, with whom Schimmelbusch is in entire agreement, there arise many new acini; isolated gland lobules show, instead of 12 to 20, several hundred single acini; there follow epithelial overgrowth in the acini, then primary cyst formation from epithelial destruction, then secondary enlargement of the cysts by confluence of smaller ones; parts of the partitions which remain jut into the cyst cavities as spurs, or in forms similar to those of papillary growths. Leucocytic infiltration to any degree is never present. Reclus, on the other hand, denies that the disease is of an adenocystic nature, but believes that it is due to a chronic inflammation. A newer theory, sponsored by Krompecher, states that the cysts are due to malformations following embryonal growth, similar to those which occur in sweat gland-cysts, *e.g.*, in the large sweat glands of the axillae. Therefore these cysts in the breast are atavistic structures returning to the phylogenetic early sweat gland nature of the breast. Peculiar "pale epithelial cells" with changes into flattened epithelium (the so-called "mosaic epithelium," which was described years before, v. Saar and others), and myoepithelial elements in the basal membrane are given as histological proofs of this hypothesis. Carcinomas with "pale epithelial cells" may arise from the cysts of this "hidrocystoma mammae." The cysts must, of course, be differentiated from retention cysts, to which latter classification belong the galactocele—see p. 1802—and small cysts in sclerotic mammae. Aschoff also believes there is a malformaton of the tissues and that the designation "mastopathia cystica" (dysplastica) is

the correct one. Pribram has developed the concept that this condition, named by him "polycystic breast degeneration," is caused by a hormone coming from the ovary, which in the senile breast produces, instead of the useful proliferation, a form of adenomatous overgrowth, cystic, adenomatous nodules and even carcinoma eventually. There is a degeneration present in the sense that the organ in senile atrophy loses its specific organic individuality, (structural equilibrium of the parts to the whole), and thus separate parts, without their former mutual inhibitions and stimuli as during the sexual period, grow, while others regress; this is the fundamental cause of the disease. Lately, the view has gained ground that the condition is neither an inflammation, nor a real tumor, but a diffuse fibromatosis of the breast due to chronic irritation, and is related to the physiological involution of the organ. The connective tissue overgrowth is said to lead to the formation of retention cysts, while, on the other hand, in addition to regressive changes, there is epithelial overgrowth of a secondary nature; infiltration foci are said to be connected with resorption processes; they may, however, be due to an inflammation, but of entirely accidental occurrence. (Lukowsky, lit., Consten, see also Bertels.)

The development of carcinoma in such breasts is very frequent. Reclus, who recognized this in his first communication, later stated that it is only an occasional coincidence, and that this disease has nothing to do with carcinoma. Furthermore, the clinical solitary cyst (see p. 1802) is considered only a form of the disease, in which the larger cysts hide the smaller ones. General experience, however, in which the author participates, seems to contradict this view (see also van Lier). When, however, Salomon says that in cystic mastitis he saw carcinoma only of the ordinary solid type and never saw carcinoma in the cysts, the author cannot agree with him (see also Ewing, lit.).

Translator's Remarks.— To elaborate further on the abnormal involution theory, McFarland has intensively studied a large number of breasts from women of all ages and under all circumstances, and finds, briefly, that acini begin to appear at the time of conception, and increase in size and number until, at the time lactation is established, the breast contains little but glandular tissue. When lactation is interrupted, involution occurs until the breast is restored to a structure containing ducts and but few acini and a number of undifferentiated-appearing round cells. There is a coincident increase in the perilobular connective tissue; choking off of ducts or acini may occur. This leads to retention of secretion and interruption in the involution of the lobule and

produces small or larger cystic structures, the so-called chronic cystic mastitis. This may be localized or generalized. If localized, a "lump" appears in the breast which is difficult and sometimes impossible to distinguish clinically from a true tumor, benign or malignant. On removal, however, since the structure is part of the breast itself, it cannot be shelled out as a benign tumor and it shows none of the hardness, infiltration, solidity,

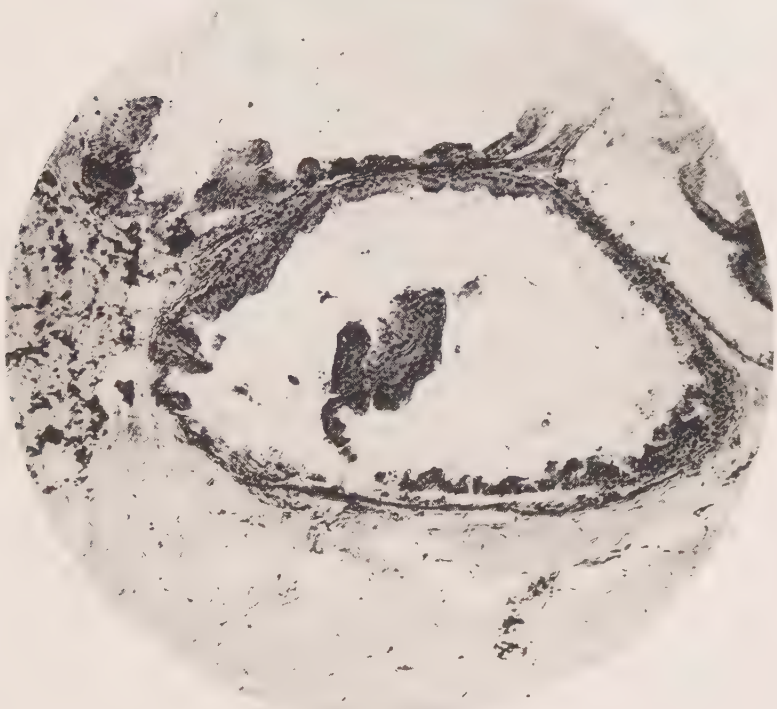


FIG. 881.—Abnormal involution of the breast showing a dilated duct lined with high "active looking" epithelium. Neighboring ducts are also shown, the partitions between them being very thin. It is readily conceivable that these partitions can rupture, leaving projecting spurs similar in appearance to papillomas but entirely different in origin. It is also readily conceivable how the contents of these duct spaces may be blood-tinged, may be thick, may be thin, may be highly cellular, etc., but also that the process, however different the pictures it produces, can be one and the same thing, namely, an aberration in the involution process. See text. L. H. #940.

umbilication, etc., of a malignant tumor. Furthermore, it contains tiny cysts or perhaps larger cystic structures. Such secondary changes as rupture of dividing walls between cysts allow small hemorrhages to discolor the cyst contents, and allow stumps to project into the cystic cavity as though they were papillary growths, etc. Such structures have been shown to remain for as

long as thirty or more years after the last lactation. It is quite possible also that at each subsequent lactation a new crop of lobules is produced from budding of the finer ducts and possibly also from differentiation of the previously undifferentiated cells. For further details, consult McFarland, Joseph, Arch. Surg., 5: 1: 1922.

In the opinion of many, inflammatory factors are more important than this involution process, but in the translator's opinion, some of the cells regarded as inflammatory, are possibly not so, and he fails to see, in many cases, any signs interpretive of inflammation. It is of course quite possible that any inflammation present is secondary to the presence of the cystic structures. The question remains to be decided; the chief difficulty arises from the fact that we cannot predict what would have happened in a breast had the surgeon not removed it, or had the patient not died. It is impossible to remove small bits every month or year or two from an individual for study of the progression of the condition, and even if it were possible, the operative procedure itself would influence the course.

In regard to the relation of carcinoma to this condition, the translator is quite skeptical, granting that it is possible but believing it infrequent. In nearly every breast removed surgically for carcinoma, or obtained without disease at autopsy, careful search will find an area or two, if not more, of abnormal involution. Also, in the removal of benign tumors from young girls, a bit of surrounding breast tissue is frequently removed with it. The translator has often seen similar pictures in these bits of tissue. The stimulus, of course, is not pregnancy in these cases, but the breasts of most women swell at menstrual periods, and now and again the breasts of many women will secrete a drop or two of clear fluid at this time.

The intracanalicular cyst adenoma (see p. 1778) is best separated from this typical chronic cystic mastitis in spite of the teaching of Tietze.

Infectious Granulation Tumors of the Breast.— *Tuberculosis* is rare, and known to occur only in individuals after the age of puberty (Reerink, exceedingly rare in males; Geissler, lit.). In six recent observations, the author observed ages of 40, 45, 46, 50, 57, 68 years. It comes most frequently via the hematogenous route with the exception of cases in which the process spreads from the neighborhood, as from caries of ribs, or from the lymph nodes of the axilla in which it is a retrograde lymphatic infection. Ascending infection through the ducts is no doubt exception-

on. These various mixed tumors are composed of a combination of epithelial adenomatous parts on which the principle accent should be laid, with a connective tissue part of such peculiar structure that it is necessary to use a double terminology to describe them (for instance, fibroadenoma). We will first discuss the fibroepithelial tumors in which the adenoma and fibroma are combined. According to the predominance of the adenom-

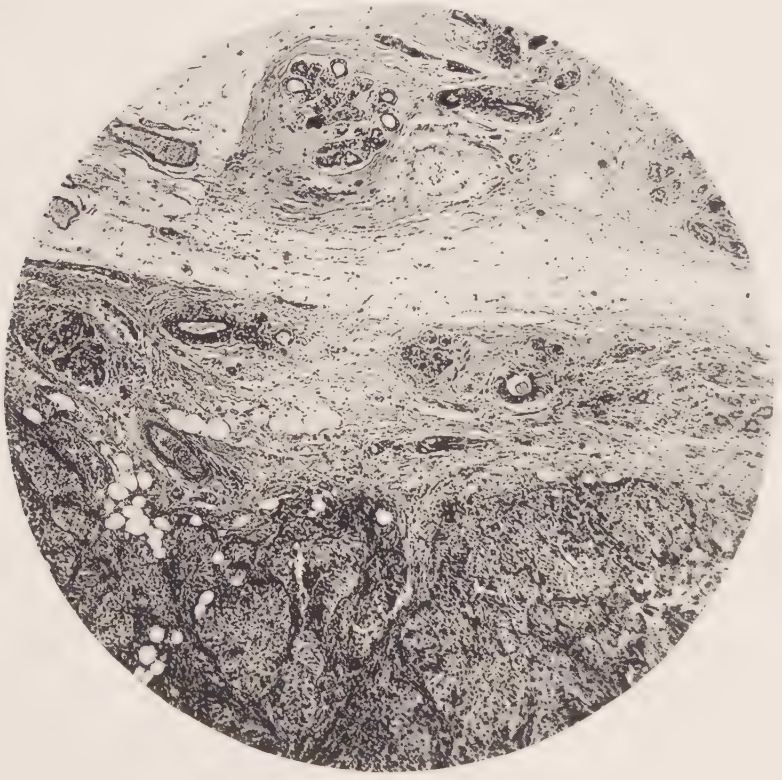


FIG. 883.—Adenoma of the breast. The edge of the tumor is shown with surrounding normal breast tissue. This patient was 28 years old when first seen, and complained of a nodule in the lower outer quadrant of the left breast, discovered two weeks before. It was painless, freely movable, not attached to the skin. At operation it could not be shelled out but was excised. Numerous sections showed a composition of large cells resembling those of the ducts, divided into alveoli by connective tissue bands. The cells were all well preserved and showed great regularity and evenness. At no point was there invasion any more than shown in the illustration. After five years, there has been no recurrence of this tumor, the patient meanwhile having given birth to a child which she nursed for some months. The first inclination was the diagnosis of carcinoma because of the fact that pure adenomas of the breast are such rare tumors and because of the fact that grossly it could not be shelled from its surroundings, but inasmuch as growth has not returned over a period of five years, it is quite probable that biologically, the tumor is non-malignant and hence an adenoma. × 60. L. H. #16777.

atous or the fibrous part, they are spoken of as fibroadenomas or adenofibromas. They appear as nodular masses as large as almonds or hen's eggs, seldom larger, often multiple, encapsulated and sharply outlined,

and thus easily shelled out from the breast with its system of lobules. Only at times are they painful; they are composed of fibrous tissue and embedded epithelial gland-like structures, and usually appear after the thirtieth year, but quite often before this, or at the time of puberty. They are very rare in males.

The point of origin, as, among others, Ribbert and Wilms assume, is from snipped off fetal parts of the gland, *i.e.*, germinal parts which contain both epithelial and connective tissue potentialities; the most important point for the substantiation of this view is the fact that they are usually plainly encapsulated. Burkard brings forth an observation for the truth of this view of congenital anlage displacement in a case in which such a tumor was found in the left breasts of each of twins. (Concerning the frequency of embryological malformations in the breast, see also Letulle.) But the encapsulation, on the other hand, may very well be due to a characteristic of the tumor growth itself, and cases can be observed in which, alongside of outspoken nodules of intracanalicular structure, there are new glandular complexes which become more and more differentiated into nodules through the addition of connective tissue and the pulling apart of the growing glandular elements. Thus Beneke believes that the adenoma is a product of the growth of the mature glandular tissue, occurring chiefly because of the entrance of ducts into the adenoma. Stoerk and Erdheim believe there are relationships to mastitic processes. The author holds this view for a certain number of the cases in which the adenomatous overgrowth is not so sharply outlined and can be differentiated from carcinoma only on microscopic examination. A total fibroadenomatous change of an entire breast (rare), in which each single lobule is changed into a small fibroadenoma and in which, as stated on p. 1771, there is marked enlargement of the breast, has been called "mastoma," or "mammoma," (see Schmincke). The author does not approve of this term, because it suggests that a tumor takes on the type of a normal breast; if this is the case the term hypertrophy or hyperplasia is better. Total fibroadenomatosis of the breast, or diffuse fibroadenoma, seem to be better terms.

We may also look upon the fibroadenomas as being adenomas in which there is coincident growth of the fibrous parts, a richer growth takes place in the "pure" adenomas, but, on the other hand, the marked active growth of glands gives to the histological picture its characteristics. Here and there are groups of glandular vesicles; in other places there are elongated, simple, branched or tree-like tubes or ducts; therefore the term fibroadenoma acinosum and tubulare have been used; usually, however, these forms are mixed (fig. 884). In one section, or even in a number of isolated sections, the tumor may show principally the acinus or the tubular

character. Fibroadenomas, very rich in glands, as illustrated in fig. 884, have also received the name of "proliferating adenomas." But from the structure of the epithelial formation and from the fibrous parts of the tissue which produce the pulling and twists of the tubules, it is plain that they are merely fibroadenomas. There do occur fibroadenomas, however, in which microscopically the principle picture is of cyst-like spaces from the walls of which pass many branched, wide tubules which, lying in the

FIG. 884.—Acinous and tubular mammary fibroadenoma; also formation of irregular small cysts; from a nodular, well circumscribed tumor of woman 36 years old (low power).

rich connective tissue, surround the spaces. These spaces with their tubules seem to be a peculiarity, a sort of pathological duct formation with pathological acini. The vesicles and the ducts in the glandular formation are lined by one layer of epithelium, or here and there by two layers, or even by more; occasionally, the spaces are entirely filled by cells. Usually, there are two layers, the lower layer composed of cubical to flat cells, the upper of cylindrical cells. Minor degrees of atypical change, without wide spread, well developed polymorphy of the cells as in carcinoma, still allow the tumors to be of benign nature. Krompecher called attention to the presence of "pale epithelium" which occasionally is

arranged in tufts surrounded by bands of smooth muscle fibers; these are quite similar in structure to the normal sweat glands of the axilla (see p. 1765). If the fibrous parts are predominant and the growing glandular parts are sparse, the tumor is more closely related to the connective tissue tumors and may then be called adenofibroma. In contradistinction to the conditions in pure adenomas (p. 1772), Kuru found in these cases, metachromasia of the pericanalicular connective tissue which acts as a new growth and which may be considered in adenofibromas as the primary factor which the epithelial overgrowth follows. Berka and Chalatow, however, doubt the significance of this metachromasia. Most of the nodular tumors which resemble fibromas because of their hardness and

FIG. 885.—Adenofibroma of breast, section through tumor shows well defined encapsulation from surrounding fatty breast tissue; recognizable cysts with wavy lines; microscopically, a pericanalicular fibroadenoma; woman 44 years old; nat. size.

their fibrillar section surfaces, are in reality adenofibromas; nodular pure fibromas are very rare.

The structure of those tumors made up of adenomatous and fibrous or myxomatous or edematous fibrous or sarcomatous parts varies considerably among themselves. Not rarely the fibrous parts develop into thick coverings around the ducts and the lobules (pericanalicular fibroma or plexiform fibroma or adenofibroma).

Microscopically, the fibrous adventitial tissue, from which the pericanalicular growths proceed, is more richly cellular than the interstitial connective tissue, and more finely fibrillar, or perhaps, in certain cases, poorer in cells and hyalinized; it can, however, be sharply distinguished from the interstitial connective tissue which, as the pericanalicular covering increases in thickness, is often reduced to mere strands carrying blood vessels. A lead pencil-like fibrous thickening of the ducts resembling the plexiform fibroma is frequent in old women. The author saw this in a 66 year old man; it was combined with cystic dilatation and atypical epithelial growth in the ducts.

Macroscopically, on longitudinal section, the surface is wavy, and on oblique section, is nodular and either fibrillar and striped, or white or glassy (hyalin). As a whole, the tumor is quite hard. Similar appearances are given by those tumors in which the pericanalicular components take on the character of fibromyxoma, myxoma or fibromyxosarcoma. It may be mentioned in passing that the term "myxomatous" is morphologic, while chemically, the structures are not myxomatous, but merely

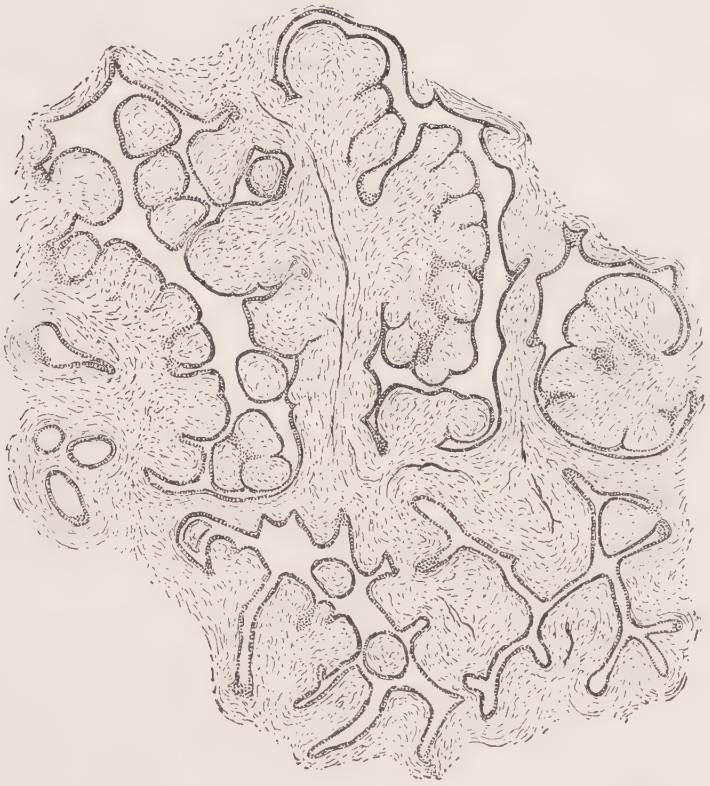


FIG. 886.—Peri- and intracanalicular adenofibroma of breast. In the distorted ducts are numerous nodular polypoid connective tissue proliferations covered by epithelium; in other places these intracanalicular proliferations appear in cross section apparently free in the lumen and surrounded by epithelium. The interstitial connective tissue is shown in some places with delicate blood vessels. From a firm nodule as large as a walnut which was shelled out from breast of girl 19 years old. Macroscopically, cross section showed finely leaved structure. (Low power.)

edematous. When richly vascularized, the freshly extirpated tumor may be bright red or dark red in color. Tumors with pericanalicular arrangement of the connective tissue may be combined with those of intracanalicular architecture, so that in one place the former, and in another place the latter type is found.

The glandular tubes of the adenoma parts of adenomatous mixed tumors are frequently rounded, but still more frequently are pulled out into elongated, irregular cystic cavities, which can be seen macroscopically as fine spaces and passages on the section surface of the nodular, sharply outlined tumor (cystic fibroadenoma).

These spaces belonging to the adenomatous parts are probably caused less by the collection of material in their lumena than by pulling by the connective tissue in the neighborhood, (according to Ribbert); but the lining epithelium grows with the connective tissue. Eventually, fluid may collect in the spaces, and finally the cysts become spherical.

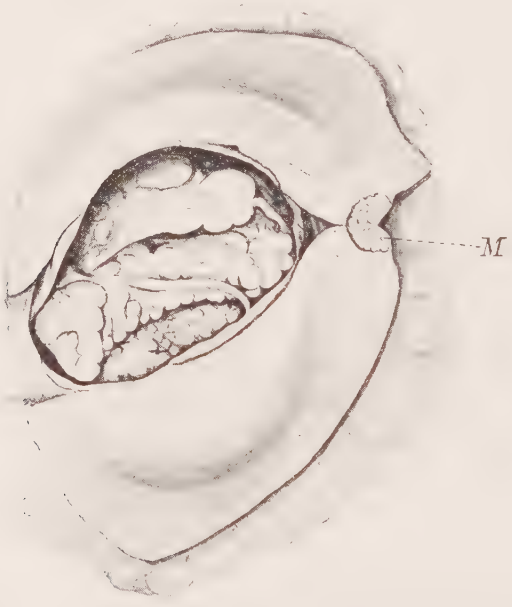


FIG. 887.—Cystic, edematous intracanalicular adenofibroma of breast; tumor opened by radiating cut, shows cyst with septa and soft translucent lobular warty tumor masses; M nipple; ⅘ nat. size.

These intracanalicular adenofibromas (myxomas—sarcomas), no doubt arise from a fibro-epithelial point, but the growing connective tissue part determines the direction and form which the entire tumor takes. It pulls apart the glands in the most bizarre forms until they finally become wide spaces, so that in the spaces are structures looking like polyps or papules or leaves or cauliflowers. All nodules and outgrowths cut obliquely or longitudinally in microscopic sections, are covered by a layer of cubical or cylindrical epithelium which has no doubt been shoved into the gland spaces by the connective tissue, but which has also actively grown into these situations. Therefore, since this tumor is an adenofibroma, the gland-like pictures on the inside of the intracanalicular growths,

as well as in their walls and in the neighborhood of the canals, indicate quite clearly that there is an adenomatous (epithelial) part to the tumor. The macroscopic section discloses that it is sharply outlined from the surroundings; it very often resembles the head of a cauliflower (Virchow), and there are many elongated spaces and larger cysts, the latter containing leaf-like branching masses pressed into their smooth walls. The arborescent structures are separated by small spaces or hang free in the

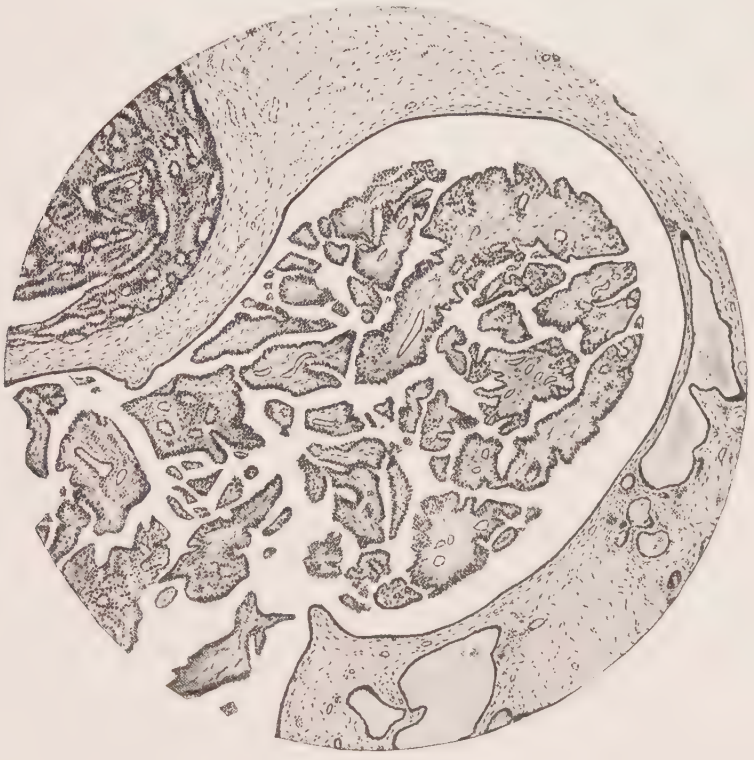


Fig. 888.—Papilliferous cystadenoma (papillary fibroepithelioma developed in cysts) of the breast; papillae covered with cylindrical epithelium (without basal membrane); to the right, a number of small cysts; to left above, a denser area filled with clefts and alveoli lined with cylindrical epithelium which does not show papillary character; woman 36 years old. (Med. magn.)

cyst. In other situations, the cysts are filled with fluid. We may speak of them as "intracanalicular fibrocystadenomas." The nodular or grape-like growths may be lifted out, even completely (oblique section), or as polyps connected by thinner or thicker pedicles to the walls of the spaces (longitudinal section). Occasionally, the number of cysts is quite small (fig. 887).

The author saw an intracanalicular adenofibroma as large as a chestnut in the right axilla of a 49 year old woman.

The more solid parts of the tumor between the cysts are, in part, fibrous, fibrillar, tendinous and in part soft, edematous or colloid.

In the sarcomas, with which myxoma and fibroma may be mixed, the consistency is highly varied; the tumor is usually soft, juicy, rich in blood, hemorrhagic, necrotic or fatty, which makes the section surface variegated and complex.

In addition to the tumor nodules, the spaces and cysts contain serous or mucoid, or thicker, milky, or pulpy yellow-red masses containing cholesterin and blood pigment. Occasionally, scattered cornifying white epithelial cells are found (cholesteatoma-like breast adenoma); carcinomatous changes have also been observed, (see Lahm), and instead of a cubical or cylindrical epithelial lining, there is an epidermoid-like lining (lit. by Grohe, Stoerk and Erdheim, Konjetzny). Such metaplasias with the formation of small pearls are also found in isolated situations of fibroadenomas.

It is not uncommon to find that fibroadenomas become stationary and disappear after undergoing hyalin changes, necrosis and calcification. The author has observed this in six tumors from the size of peas to chestnuts; the bearers were mostly older women, (in four cases 60, 63, 74, 79 years).

The intracanalicular connective tissue tumors of the breast usually grow slowly, but the type of tumor may change and the poorly cellular fibromatous parts take on a richly cellular sarcomatous nature; as this component becomes dominant, the growth is much more rapid. Thus cases have been seen in which the benign extirpated tumor recurred after a short time (see case of Prym), and then produced sarcomatous metastases (see lit. by Gorham). Tumors weighing twenty and more kilos have been reported. Even the arborescent sarcomas occurring more frequently in young women, usually grow slowly, and as long as they have their localized outline, are painless and disturb the general welfare of the patient little, or not at all; when they occur before the thirtieth year, have grown slowly and are extirpated, they usually do not recur. But, intracanalicular sarcomas arising in later years recur repeatedly, and even though seldom, may be the point of origin of distant metastases. Not rarely, the walls of neighboring cysts are destroyed by pressure and the remnants of the septa can be recognized in the larger cysts (fig. 887). After pressure necrosis of the cyst wall and the external skin, these growths, especially if soft and richly cellular like myxomas, myxosarcomas or sarcomas, may grow through the skin to the surface of the breast, and appear as lobed, polypoid or cauliflower-like growths with a tendency to suppuration. They give a first impression of malignant infiltrating tumor, but the cyst cavity can be entered by manipulating a probe through the tumor masses;

it can also be determined that the skin is not infiltrated in the immediate neighborhood of the rupture. Such outgrowths occur in large as well as in relatively small tumors. The intracanalicular growths occasionally grow along the excretory ducts until they appear in the region of the nipple; their surface may eventually be covered by squamous epithelium. Different names have been given to them, "cysto-sarcoma," or "cysto-sarcoma phyllodes" (this term means flat; by it are understood many histologically different tumors), "fibroma-myxo" or "sarcoma proliferum" or "arborescens" (J. Muller), "fibroma (myxoma, sarcoma) papillare" or "polyposum intracanaliculare." "Cyst-adeno-papilliferum" (the so-called intracanalicular cyst adenoma, see Sasse) or "cystic papillary epithelioma" (more strictly fibro-epithelioma), are

FIG. 889.—Rupture of a cyst-sarcoma phyllodes through the skin of breast; to the left in the corner, the nipple; ¾ nat. size.

names which are best given to tumors with capillary excrescences growing from the walls either into preformed canals (milk ducts) or into newly formed glandular spaces and cystic cavities; these are found usually in young girls (author, 18 year old; Nagel, 14 year old) and younger women. In their differentiation from the papillary growths of the ordinary fibro-adenomas with intracanalicular connective tissue overgrowth, it should be noted that their fibrous parts are usually predominant, their papillae have a tender fibrous stalk, and the thinnest papillae, in contradistinction to the two celled layer of the adenofibroma, are covered in most situations by one layer of a polymorphous cylindrical epithelium which has no membrana propria. In one case, resembling a carcinoma, Buday found ciliated epithelium. The tumors usually appear as multilocular cystic growths in which the cysts, stretching and thinning the skin, can be seen shining through; from the inner wall, usually smooth and pale, although

occasionally brown or red in color, are found, here and there, soft masses which microscopically have an exquisite papillary structure (fig. 888). In a 45 year old woman, the author saw a cyst grow rapidly after having been stationary for many years. There are also cases, or situations in other cysts, in which the cells are present in alveolar or cylindrical architecture, and the papillary cystic form is not so obvious, at least macroscopically. Clinically, the "bleeding nipple" is characteristic, as the author has seen in a 35 year old and a 50 year old woman (see the literature by de Josselin de Jong). They are very similar to the papillary cystic adenomas of the ovary (p. 1561). Here and there the benign papillary growth may become carcinomatous (fig. 902), but still remain similar macroscopically to the benign cysto-papilloma (see also Pusateri, de Josselin de Jong, Jacoulet, lit.). Papillary cylindrical stalks, other epithelial structures, as well as papillary carcinomas, may arise even from the inner surface of solitary cysts the results of dilatation of the ducts (the author observed this occurring to the external opening of the ducts, see Brandes, Cornil, Fremicourt). These can be called briefly "encysted papillomas and carcinomas." Carcinomatous transformation in these fibroadenomas (see de Quervain, Soloweitschick, Knapp, lit., Jaffe) has been observed by the author in a case published by Kuru (44 year old woman, tumor observed for one year; four months previous to extirpation it had grown very rapidly); epithelial cells leave their typical organic connection with the adenomatous gland structure, grow atypically and infiltrate the surroundings (see p. 1789).

Sarcomatous transformation (the so-called sarcomatous degeneration) in adenofibromas or adenomyxofibromas of the breast occurs, but is comparatively rare (lit. by Gorham).

The author has repeatedly seen fibroadenomas with carcinomas; in one case (31 year old woman) the small fibroadenoma lay within a carcinoma.

Pure Aglandular Tumors of the Connective Tissue Group.— By these are understood tumors in which there is no new formation of glands in the actual tumor. They are rare, speaking generally, particularly the benign varieties; among them are fibromas, present as diffusely spreading, or as circumscribed, occasionally multiple, hard, lobulated masses, cut with difficulty, occurring about the nipple, or, as the author has seen, as pendulous affairs. Atrophic glandular structures are perhaps recognized microscopically. Myxomas or fibromyxomas also occur, as do myomas or myofibromas; the author saw a fibromyoma of the nipple as large as a hazel nut in a 40 year old woman, (lit. by Bauer); cavernous hemangiomas are unusual and, as a rule, diffuse, but more or less encapsulated. The author saw such a tumor as large as a lentil on the nipple of a 14 year old

girl (lit. by Marangoni, Carl). Enchondromas are rare; osteomas and osteochondromas have been observed (Cornil).

Infrequently, small osteoid or hyalin cartilaginous masses (metaplasia) can be found in cyst-adenomas, sarcomas, and carcinomas. Davidsohn recommends cresyl violet as the best dye to demonstrate cartilage; it stains light red. Lipomas of the breast are sharply outlined, soft or elastic, or pseudofluctuating tumors in the breast itself (rare), or diffuse hyperplasias of the fatty tissue; those which occur subcutaneously in the

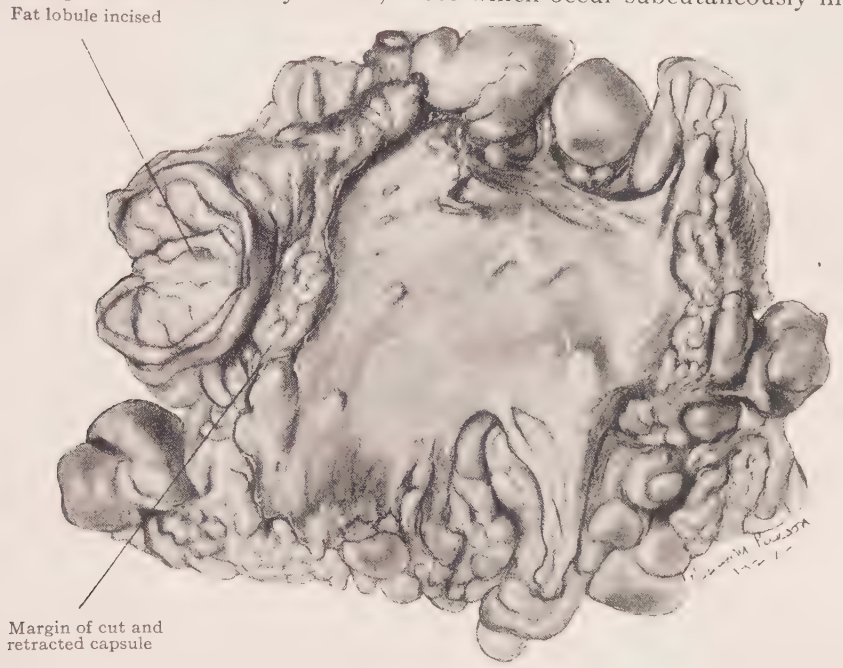


FIG. 890.—Lipoma of the male breast. This is a rare tumor in our experience. Was present in a man 42 years old for 10 years. It consisted of a number of distinct lobules of fat, each surrounded by a very thin delicate connective tissue capsule. The whole is shown. The fat has been peeled out of the largest of the lobules. No recurrence in 1 year. L. H. #718. 5/8 nat. size

region of the breast or its neighborhood or behind the breast may be designated as paramammary lipomas (Rouvray).

Solid sarcomas are somewhat more common. They occur as nodular, large, quickly growing, diffuse tumors, the most common of which is the small round cell sarcoma. Sarcomas may be bilateral. In contrast to the so-called sarcoma phyllodes (p. 1781), these behave as true malignant tumors. They infiltrate the subcutaneous tissues and the skin, which is no longer movable and gradually disappears as fungoid nodules appear on the surface. The tumor may then undergo softening. Occasionally the tumor infiltrates below the breast into the pectoral muscles, the ribs and pleura. Cachexia and distant metastases occur, just as in any other

rapidly growing sarcoma. The lymph nodes in many cases are not involved (Gross). Etiologically, traumata are said to play a role.

Lit. concerning sarcoma by Finsterer; concerning breast sarcoma in the male by Mitterstiller; see also Mohnle, Scheller.

There also occur enormous, quickly growing, round cell sarcomas, soft, juicy, pale red and of uniform histological structure. A smear preparation will show free nuclei. Furthermore, there occur combined round and spindle cell sarcomas, as well as the relatively frequent pure spindle cell tumors of tough consistency, occasionally with a regular radiating arrangement of the fibers. In a 62 year old woman, the author saw a fascicular, richly vascular, spindle cell sarcoma growing from the brown, smooth, inner surface of a cyst as large as a mandarin orange. It contained many giant cells. It is uncommon to see mucoid pseudocystic softening, but in a 49 year old woman with the clinical diagnosis "cystic mastitis or carcinoma," the author saw a richly vascular spindle cell sarcoma with giant cells, which had softened into a cyst as large as a hen's egg. There have also been observed polymorphocellular sarcomas with thick cells, often with giant cells. In a 43 year old woman, the author saw a tumor as large as a child's head in the lower outer quadrant of the breast, which had appeared during pregnancy and had reached an enormous size in four weeks; it was lobulated, on section glassy, gray-red and composed of polymorpho-spindle cellular tissue. Such tumors seem quite soft, fluctuating, and tear easily because of focal myxomatous or fatty softening, necroses and hemorrhages with pseudo-cyst formation; these false cysts may reach great size (see below); one as large as an apple was seen by the author in a 61 year old woman. There may be present isolated true cystic dilated ducts. In other sarcomas, in addition to the polymorphous or spindle cells, there are so many giant cells that the name giant cell sarcoma is justified, (a number of them take on an alveolar arrangement). The author has examined such cases which grew shortly after infliction of trauma. Others are angiosarcomas, usually perivascular, (p. 1670), but telangiectatic sarcomas also occur. v. Haberer-Pommer described a leiomyoma, probably arising from the muscular elements of the arterial vessels in which there was sarcomatous growth; this tumor occurred in a young man. Furthermore, there occur alveolar sarcomas, and even melanotic forms of these, and round cell sarcomas containing cross striated muscle fibers. There are also endotheliomas arising from the endothelium of the vessels. Arnold has described a chondro-sarcoma. The author saw a chondro-sarcoma weighing 760 grams arising in a lobulated cyst; the structure was found in a 57 year old woman who had noticed the tumor for 27 years; for the past half year it had grown much more rapidly. Osteoid sarcomas (Stilling) as large as a child's head have

been observed in solid or in cystic forms, the cysts containing blood; the author observed such a case in a 46 year old woman; an osteochondrosarcoma (Sehrt, lit.) was observed by the author in a 52 year woman; this tumor is exceedingly rare.

Both sarcoma and carcinoma have occurred separately in the same breast (see *e.g.*, Schlagenhaufer); more rarely they occur as a mixed tumor, a sarco-carcinoma (Dorsch, Orth, Coenen, Takano and lit. in appendix); the author has seen such a tumor as large as a cherry, softened in the center and sharply outlined from the rest of the breast. In a second case of

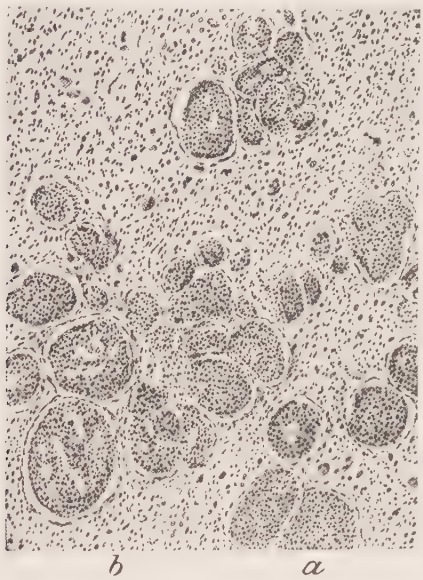


FIG. 891.—Carcino-sarcomatous area from a chondrosarco-carcinoma of breast of woman 63 years old (see text); a, carcinoma nest; b, sarcoma tissue with many giant cells. (Med. Magn.)

carcinoma sarcomatodes (Takano), the type was that of the giant cell carcino-sarcoma of Krompecher with a very small amount of cartilage; in a third case occurring in a 45 year old woman, medullary carcinomatous masses were less sharply outlined from the spindle and giant cell sarcomatous tissues than in the two previous cases. The few observed cases have all shown sharply outlined boundaries from the rest of the breast. Another case of the author is unique in that he observed a pseudocystic chondro-sarcocarcinoma which had reached the size of a man's head; through destruction of the central part it had turned into a thick walled cystic mass; it was removed from a 63 year old woman (fig. 891); the observation of Waelle was similar to this case.

Metastatic sarcomas of the breast are very rare (lit. by Plew).

In the differential diagnosis from giant cell sarcoma, there must be taken into consideration: (1) aleukemia, (2) diffuse leukemic or chloroleukemic infiltrate, (see p. 273, the observation of the author as well as Simon, Mont Reid, McWilliams and Hanes); (3) lymphosarcoma, usually coming from the mediastinum (Schoen, observations in gynecomastia, and lit. Fabian).

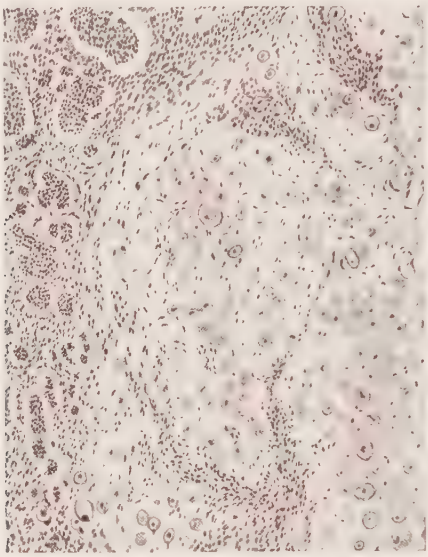


FIG. 892.—Predominant cartilaginous area from the chondrosarcocarcinoma of breast in fig. 891; to left, and partly between the cartilaginous masses are sarcoma and carcinoma. (Med. magn.)

Carcinoma of the Breast.- Carcinoma is the most malignant and the most frequent primary tumor of the breast. This is particularly true of the female breast, which, according to Billroth, is involved as a rule between the thirtieth and the sixtieth years, seldom earlier, (17 years old in an observation of the author), or later into the tenth decade; it usually appears in otherwise healthy women, and in those single as well as those married. Even in males, carcinoma is the most frequent of breast tumors. [For carcinoma of the male breast, see Wainwright J. M. Arch. Surg. 14: 836: 1927.—*Translator.*]

According to Deelman, it is more frequent in married women, during the child bearing period, and more frequent in single women after this period. Pregnancy and lactation favor the growth of breast carcinomas (see, among others, Wolff).

The relative proportion of male breast cancers among breast cancers in general is 2% according to Schuchardt; according to Schultze, (3329 cases), 1.39%; see also Scheller.

There seems to be an hereditary tendency displayed by these tumors, especially in certain families.

Secondary breast cancers occur most frequently from metastasis or direct extension of a carcinoma via the lymph stream from the other breast; very rarely, a breast is involved through the retrograde lymphatic route, *e.g.*, from a carcinoma of the stomach. Hematogenous metastases have not been observed with certainty (Montier and Marre, lit., Schottlander, Silberberg).

Carcinomas of the breast arise from the acini or from the ducts, as a rule, in the interior of the organ.

The author has often assured himself that this development took place in many places at once (pluricentric). A carcinoma also occasionally arises outside of the breast proper from a misplaced fragment of breast (see also Williams).

The nipple is a very rare point of origin. When the tumor does arise here it comes from the skin of the areola or the nipple itself, and is consequently a squamous epithelial tumor; it may infiltrate deep into the breast tissue or remain superficial. Or, it comes from the larger ducts and is a squamous cell epithelioma, cylindrical carcinoma, or occasionally a small-cystic papillary adeno-carcinoma with a neatly arranged structure; the author has seen such a specimen coming from the nipple of a man. Papillary fibro-epitheliomas of benign nature and coming from the ducts in the region of the areola are quite rare.

"Paget's disease of the nipple" begins with a long lasting, itching, eczematous skin disease around the areola, which then spreads concentrically into the neighborhood. It is usually found in women between 40 and 60 years old. Around the gradually disappearing, shrinking, retracting nipple appears a bright red, glistening, superficial ulceration which spreads slowly along the surface. Its further course may be halted at times, and epithelialization begins here and there; the author knows of a case occurring in Breslau in an 85 year old woman in whom the ulceration extending over 20 years had covered the entire surface of the thorax; a similar case was described by Vignolo-Lutati. In another form, occurring in the region of the nipple or the breast itself, there appear voluminous masses of tumor tissue with the structure and the course of an ordinary carcinoma of the breast, (*e.g.*, the case of Krogius, which ended fatally in eight months). In the epithelium of the reddened areas are found striking, light "round cells," "Paget cells," at first considered by Darier, Wickham and others to be sporozoa, the cause of Paget's disease, but which then were called peculiarly swollen edematous epithelial cells (Unna), *i.e.*, cells from the epidermis itself, (Hannemuller and Landois, Gaarenstroom). As newer observations emphasize, they are, however, degener-

ated carcinoma cells which infiltrate into the epidermis (Jacobaeus, Ribbert, Hirschel and others). Pathologically anatomically, the cases are not uniform, but clinically they tend to be more so. While many believe it is a growth on the surface similar to the rodent ulcer (see under carcinoma of the skin; Karg, Eschlenoff), Depage discovered that the disease began under the areola, and then spread into the ducts as well as along the surface, finally infiltrating the breast itself; the epidermis shows overgrowth and degenerative changes. The most recent work shows that the point of origin is in the ducts (Schambacher, Dietrich), or perhaps the parenchyma of the organ itself (Kyrle, Elbogen, lit., M. B. Schmidt),

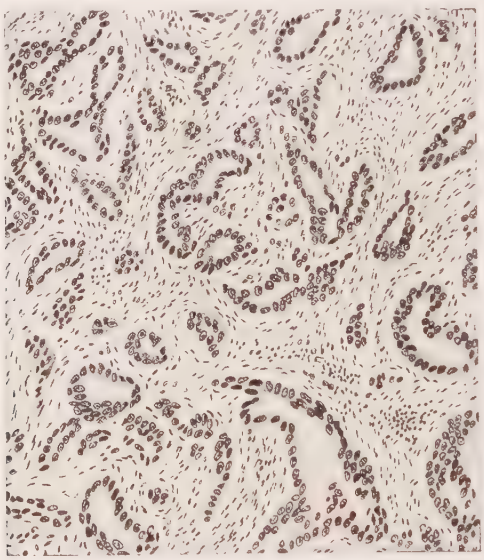


Fig. 893.—Adenocarcinoma of breast from a woman 46 years old. (High power.)

from which the polyhedral carcinoma cells soon infiltrate in and below the epidermis, which gradually undergoes destruction. Further intra-epidermal growth determines the picture of Paget's cells, and in this stage it is first observable clinically as a typical entity. The marked reddening is caused by hyperemia of the cutis. The author verifies these statements on the basis of his own observations. (See also Herzfeld, Zieler, Reuter, Kreibich.) For Paget's disease in situations other than the breast, see the lit. by Hartzell.

Histogenesis of Breast Carcinoma.— Carcinoma may arise in a previously healthy or previously diseased breast (compare pp. 1763, 1769 and 1782). According to Langhans, carcinomatous change begins in the gland acini, the epithelial cells of which begin to grow, enlarge, and infiltrate irregularly from their position into the neighborhood; the cells

fill the lumena, and the acini as a whole become thickened. Thus far the tumor is similar to an adenoma. Then the membrana propria is ruptured—previously, however, the muscular basket-cell layer is destroyed (see Letulle),—the carcinomatous cells pass into the connective tissue spaces of the neighborhood and fill them with cohering masses of cells. For these reasons, and because the gland acini take on quite irregular forms, both the origin from glands and the similarity to the adenoma

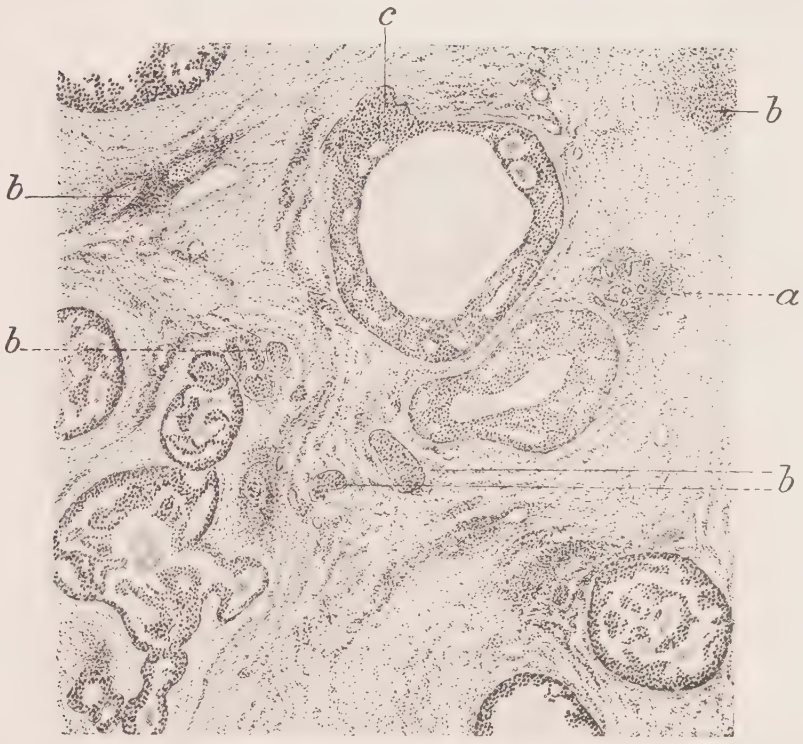


FIG. 894.—Squamous cell carcinoma of mammary ducts; marked small cell infiltration of the tissue, which shows at b, isolated solid nests of carcinoma (stratified epithelium); in other sections larger groups of solid alveoli filled with squamous epithelium; at a, group of normal gland acini with vicinity markedly infiltrated; at c, obvious invasion into the surroundings. (Low power.) From a soft juicy tumor the size of an apple; from the section surface numerous small droplets of milky fluid could be squeezed out of small holes; larger ducts dilated with cloudy, sometimes grumous yellow white contents; woman 58 years old; axillary nodes involved (solid nests of squamous cells).

disappear. While the masses of cells are usually solid and are composed of merely cuboid cells (solid carcinoma), occasionally, the tumors contain higher or lower cuboid or cylindrical cells arranged like tubules with lumena (adeno-carcinoma, see fig. 893). But these formations occur in other solid carcinomas and do not influence the macroscopic picture as in a cystic adenocarcinoma (p. 1798). If the excretory ducts are the points

of origin, the epithelium of their walls is piled up in many layers, the cells become polymorphous or large and flat; they infiltrate the neighborhood in alveolar complexes with cornification, but relatively seldom with pearly laminations in their centers. The septa following or surrounding the masses are usually infiltrated by small cells, among which, the author has repeatedly seen almost exclusively plasma cells. These relatively infrequent flat cell carcinomas of the breast, (Troell and Calderara describe two cases), are quite different from the ordinary breast carcinomas arising in the epithelium of the acini. In the inside of the carcinomatous ducts are complicated bodies, some flat against the walls in the form of broad rings around spaces, and some filling the wide lumena; other honeycomb-like cell masses are necrotic in their centers (fig. 894). On section, this form usually shows macroscopic "alveoli." It must be clearly distinguished from certain benign epithelial overgrowths of the ducts, in which the cells take on a squamous character and surround the ducts as a thick layer, or as a more or less caseous mass, without, however, infiltrating into the surroundings; this picture is occasionally seen in fibroadenomas, but much more often in "cystic mastitis" and naturally is of great importance in the differential diagnosis from carcinoma. If a carcinoma arises from an adenoma or a fibroadenoma with tissue spaces, the development can be seen in the acinous parts (p. 1782), as well as in the small cysts; the latter appear like carcinomatous ducts, while the former show much polymorphy of the epithelium. The cells stain irregularly, are piled up in heaps which often have pseudo-lumena. In the neighborhood of luxuriant carcinomatous growth, the stroma is always infiltrated by small cells, among which the plasma cell may be the dominant individual. The lymph channels are often quite obvious, not only those in periacinar positions, but also interacinar, and further spread of the carcinoma takes place through these channels; indeed isolated cells may be carried in the lymph stream to the neighboring lymph nodes very early in the disease, the earliest in the soft, richly cellular varieties. Not rarely, the cells spread into the soft pericanalicular connective tissue in strands. They may also break into the lumena of the ducts and spread in this way, while the duct epithelium is compressed by the carcinoma cells or is undermined, and lifted off, and then compressed. These pictures must not be mistaken for collections of fat-containing cells seen so frequently in normal breasts; Ritter calls the latter "colostrum bodies." Rupture into veins can often be detected in the primary tumor (Goldmann); it very probably will lead to early metastasis, *e.g.*, in the skeleton.

Histology of Breast Carcinomas.—The most frequent form arises from the glandular parts, is composed of solid masses, and may be divided into three types as manifested by a varying composition of interstitial sub-

stance and carcinoma bodies; it is understood that the types are frequently combined.

In addition to an ordinary form, the more infrequent flat cell carcinoma solidum, as described on p. 1790, is also encountered in alveolar arrangement.

Solid carcinoma simplex consists of fairly large alveoli separated by moderately thick fibrous septa, the alveoli filled with closely packed, rounded, or polymorphous, sometimes large, sometimes smaller, epithelial cells (fig. 895); glandular figures are usually lacking. Groups of these cell nests are called carcinoma bodies, and are the most important parts

FIG. 895.—Carcinoma of breast; below, carcinoma simplex; above, carcinoma scirrhosum; mitoses in cells (high power).

of the tumor. The septa are merely the supporting framework into which the carcinoma cells grow; this supporting substance grows but moderately and reacts with round cell infiltration.

Occasionally, the cell nests are elongated, (tubular carcinoma, Billroth), whereupon the tumor is harder and more infiltrating in its growth; in other cases, the nests are arranged more in the acinous forms, (acinar carcinoma, Billroth); then the tumor, as a whole, is softer, and has a nodular structure. But both forms are often combined. The excretory ducts occasionally show the growths as described on p. 1790. In a predominant solid carcinoma, there may be occasional "adeno" structures (p. 1789) in the primary tumor as well as in regional nodes; pure adenocarcinomas, on the other hand, are not very common, but according to Salomon, are said to have a relatively good prognosis. (Concerning fat

and Altmann's granules which are found in much less number than in the functioning glands, and concerning the specific function of the epithelium of breast carcinomas, see Saltzmann.)

Solid Medullary Carcinoma.— The stroma (septa) is tender, and infiltrated by small cells. The alveoli, *i.e.*, the isolated nests of carcinoma cells may be large or small, but have relatively much more mass than the stroma; the cells are rounded or angular and frequently quite small.

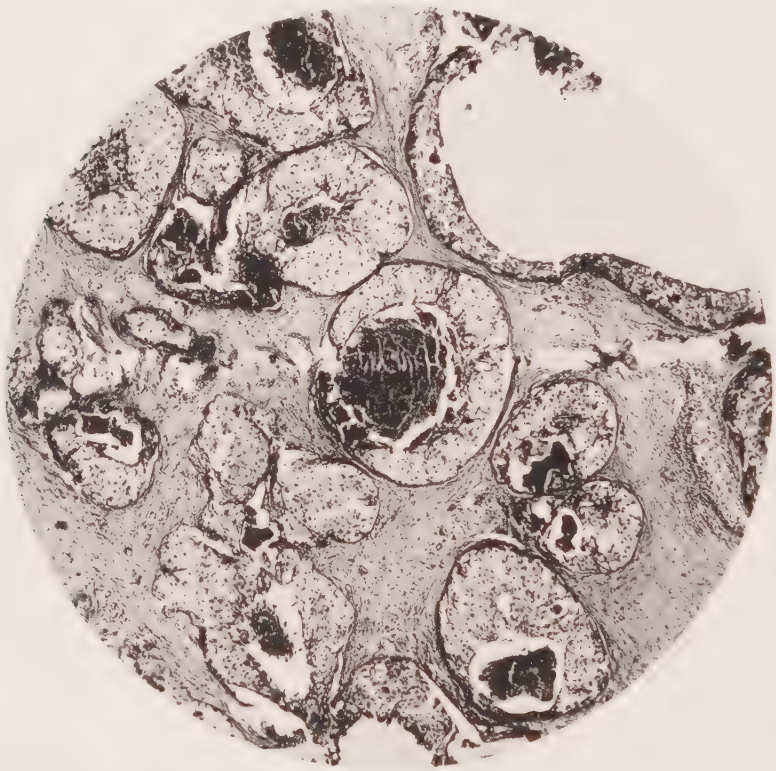


FIG. 896.—"Comedo" type of carcinoma of the breast. So-called "comedo," because grossly the plugs of debris as in the center of one of the carcinoma bodies can be expressed like a "comedo" as a long soft yellow cylinder. These are said to grow comparatively slowly and produce metastases comparatively late. × 60. L. H. #19383.

The boundary line between the tumor and the healthy parts is usually infiltrated with round cells; when similar infiltrations gather in the smaller septa and the periphery of the carcinomatous masses, the boundary line is indistinct (carcinoma granulosum).

The ducts may be lined by many layers of irregular, polymorphous epithelial cells, and capillary-containing connective tissue strands grow from their walls. They may also be filled completely by carcinoma cells, so that enormous acini are produced which undergo central necrosis.

Scirrhous carcinoma solidum, atrophic cancer, scarring cancer, is the most frequent form. Compared to the stroma the carcinomatous areas are relatively small; they infiltrate as small pointed rows of cells into the spaces of the connective tissue. Occasionally, isolated cell nests reach considerable size in the massive, often hyalinized, connective tissue (large alveolar scirrhus). In other places, the carcinoma bodies undergo complete destruction by atrophy or fatty degeneration, and nothing is found

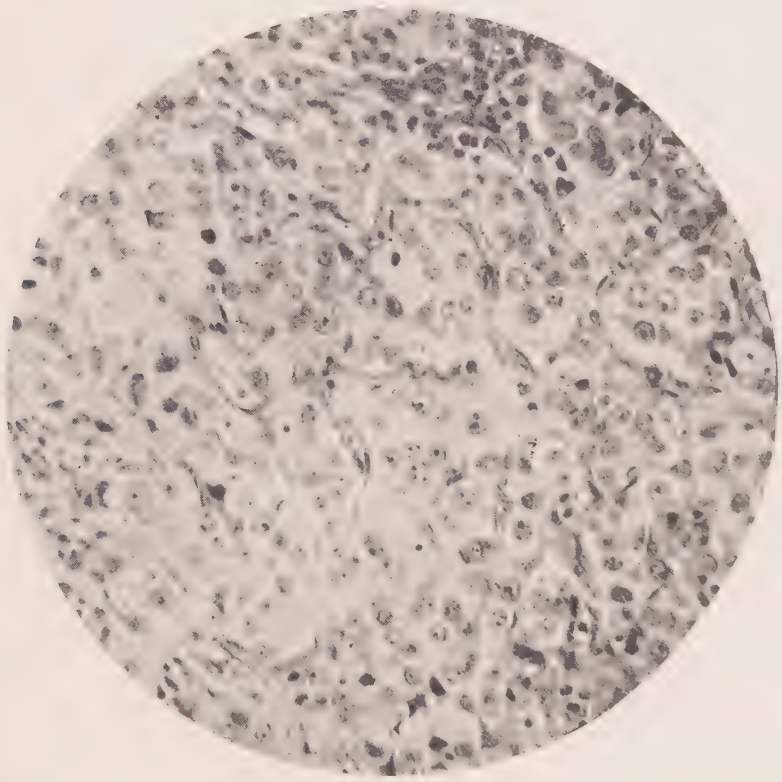
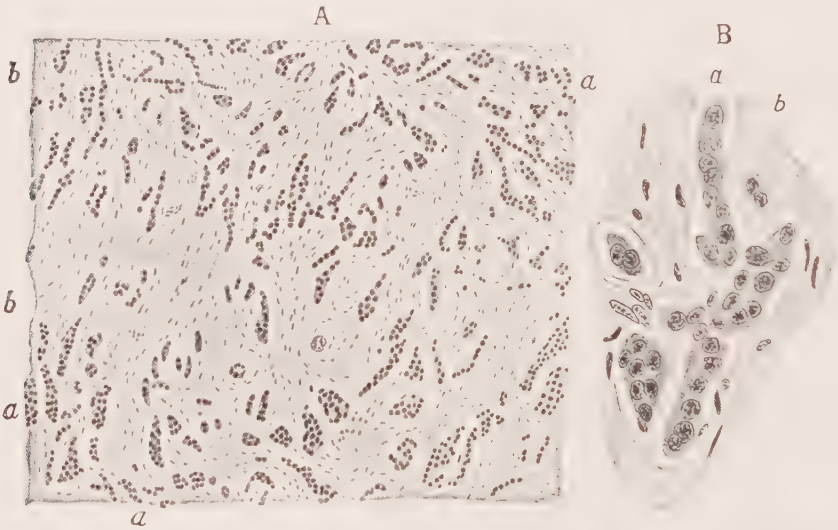


FIG. 897.—Flat, almost squamous cell carcinoma of the breast. Such pictures have given rise to numerous controversies as indicated in the text. In the translator's experience, they are met fairly frequently. × 110. L. H.

except fibrous scar tissue, in which a few remnants of the carcinoma cells remain. But in the peripheral parts, the true character can be recognized because the growth of connective tissue is still slight. Occasionally, the lymph node metastases, however, show an adeno structure. It is particularly in cases lasting several years, that the breast tumor is composed only of hyalin, sclerotic connective tissue masses with a few small islands of carcinomatous infiltrates undergoing atrophy.

Little can be added to the description of the macroscopic appearance of these three types which very frequently are combined in one tumor.

The medullary carcinoma is pale, on its section surface gray-red to gray-white or gray-yellow, and soft masses of cells (carcinoma milk) are expressed by pressure. An alveolar arrangement can often be made out, and the central parts of large acinous-like alveoli may be yellow and softer than the gray-white peripheral parts, so that a very neat glandular picture is produced. The tumor sometimes develops large, soft nodules, but in other cases there is diffuse infiltration. The scirrhus, the most malignant form, is as hard as scar tissue; the ducts may sometimes be differentiated as cloudy yellow-gray strands; in the neighborhood are usually readiating nodules, scar-like masses, or flat thickenings. Carcinoma simplex produces a picture between these two. Combinations of the different types produce pictures which cannot be definitely classified.



FIGS. 898 and 899.—Carcinoma solidum scirrhosum of breast; small solid nests (a) in abundant connective tissue stroma (b); A, with low —, B, with high power. The polymorphy of the carcinoma cells can be recognized; gaps around the nests are artefacts caused by shrinking of tissue; woman of 50. (Hem-alum-eosin stain.)

Consult Salomon concerning the clinical aspects.

According to Iselin, medullary carcinomas offer the best prognosis for operation and x-ray treatment, and next to them the carcinoma simplex; the worst prognosis is offered by the scirrhous variety. See p. 1791 concerning the relatively good prognosis in adeno-carcinoma.

Regressive Changes in Carinoma. The Rarer Forms.—Fatty degeneration is very frequent in carcinoma cells and may lead to their disappearance. The connective tissue supporting structure is hyalinized, but calcification is rare. Laminated calcification inside of carcinoma bodies is also infrequent; nevertheless the author has seen psammomous

carcinomas with calcification even in their metastases (see Neugebauer, F. J. Schmidt and others). Colloid change is found occasionally, usually in a small cell or a round cell solid carcinoma and more rarely in an adenocarcinoma; fatty changes are, as a rule, coincident, and the supporting substance may also undergo colloid change. (In the statistics collected by Lange (lit.) from thirteen sources, there were seventeen colloid carcinomas or .93% among 1814 breast carcinomas; in 2944 breast carcinomas collected by Gaabe there were 49 colloid carcinomas or 1.66%;

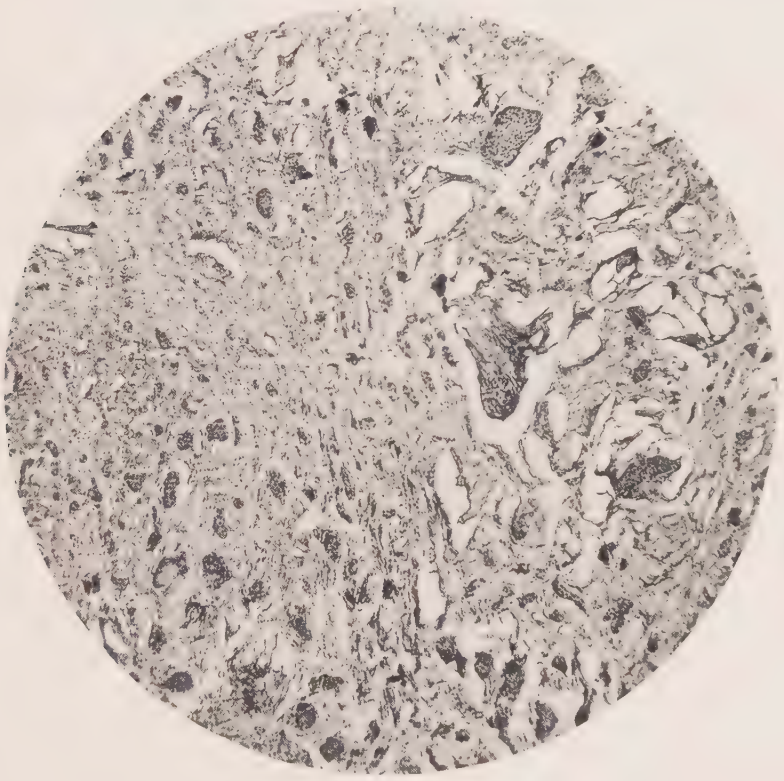


Fig. 900.—Colloid or mucoid carcinoma of the breast. From a woman 32 years old. Duration six months. Entire tumor about 5 cm. in diameter. Very early metastases in the axillae. Patient living and well three years after operation. × 60. L. H. #19236.

among 638 in the Pathological Institute of Göttingen from 1906 to 1919 there were 20 colloid carcinomas or 3.13%; among those 20, 14 appeared beyond 60 years of age; all were in women; Spangenthal, lit.) According to Lange, the colloid change in the stroma is in reality more important, while the change in the carcinoma cells themselves is not colloid, but simple atrophy and fatty degeneration. The author has studied cases in which this appeared to be true, and differentiation may be quite difficult; in most cases, however, it is probable that the colloid material comes from

changes in the carcinoma bodies themselves and merely infiltrates the connective tissue. Ribbert believes this is the only mode possible; Schmittmann, Ewing, admit both possibilities (see Sapotschinska, Spangenthal, lit.). Colloid carcinoma (gelatinosum, mucosum) grows slowly and occurs in both infiltrating and tuberous forms; in the latter type the nodules are intimately connected with the surrounding tissue and new small nodules grow from the periphery. The nodules are rounded or cake-like. In typical cases, the section surface shows a peculiar colloid appearance; there is a network or honey-comb-like structure filled with

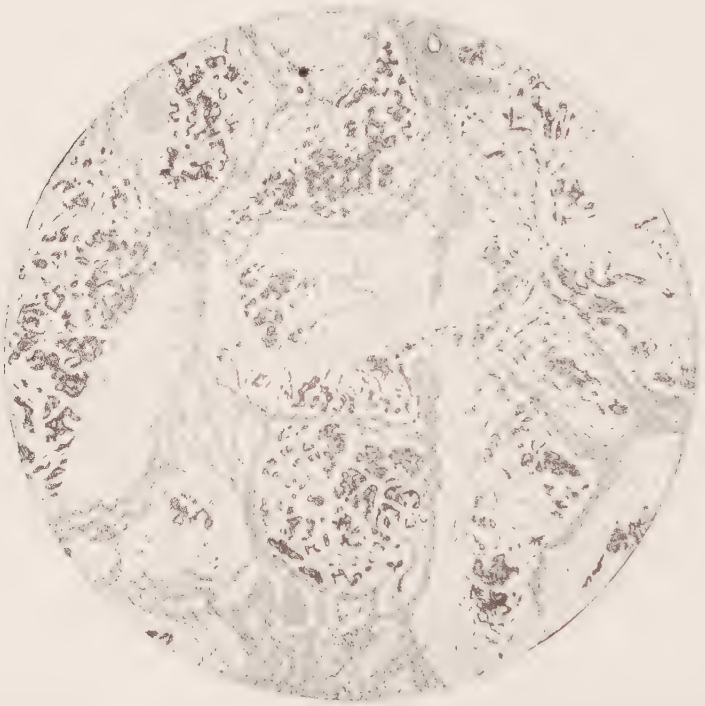


FIG. 901.—Mucoid carcinoma of breast, woman 68 years old; the carcinoma bodies show many light areas due to mucous degeneration; others are dissolved in the mucus; the interstitial tissue infiltrated with mucus and many of its fibres disintegrated. (Low power.)

colloid, and not merely a softened mass. The translucent colloid may appear gray-white, or yellow to brown, or like raspberry jelly, or spotted with blood; occasionally it may resemble a colloid goiter. The author has also observed cases in which, in addition to an ordinary glassy pale-gray scirrhous carcinoma which retracted the nipple, there was an egg-shaped colloid growth on the surface of the breast; in another case (56 year old woman) the colloid change in a carcinoma solidum was first recognized microscopically. It seems that the colloid type prefers older individuals (in 24 personal cases the author observed ages of 34 which is

abnormally early, 37, 44, 48, 54, 54, 56 years, 8 cases in the seventh decade, 7 cases in the eighth decade, and 2 cases of 82 and 83 years).

The axillary lymph nodes, which remain free of metastases for long times, may show an ordinary solid carcinoma or a trace of colloid metamorphosis; in other cases, the lymph node metastases are entirely colloid, as in the case of the above mentioned 34 year old woman. If operated upon early, colloid carcinomas have a relatively good prognosis (see also Gaabe). In neglected cases, ulceration and the formation of distant metastases occur. But the author saw two cases (83 year old and 79

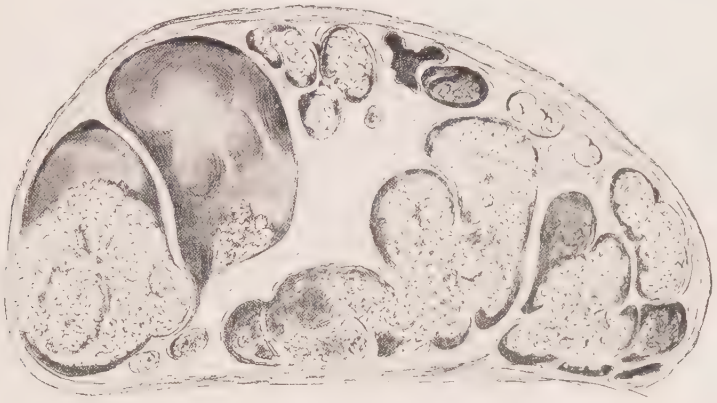


FIG. 902.—Cross section of a papillary cystocarcinoma of left breast of virgin 45 years old; duration about 3 years; infiltration of axillary nodes; ¾ nat. size.

year old women) who had the tumors for ten years; in both, the nodules were as large as hen's eggs but fairly well outlined; in the second case the lymph nodes of the axilla were infiltrated.

In many of the tumors, there are microscopic small-cystic dilated tubes filled with colloid or butter-like contents (cystic carcinoma, Waldeyer); in other cases, the tumor masses are everywhere composed of cylindrical canals, in which are microscopic finely granular or homogenous clumps of cell products, (cylindromatous carcinoma). In microscopic cross sections of larger growths there may also be seen window-like or sieve-like openings (fig. 894), so that the cell masses appear like nets; these broken up masses are everywhere in some tumors. Most tumors of this type come from the ducts and are composed of flat epithelium, but others arise from the glandular epithelium; there are even cases in which the glandular formations are so well marked as to justify the name "cylindromatous adenocarcinoma." In isolated places, particularly in carcinomatous ducts, similar pictures are found in otherwise solid carcinomas.

Other rare forms may be described as "cystic carcinomas" or "cyst adenocarcinoma simplex." These are adenocarcinomas with large

macroscopic cystic spaces lined by many layers of cells, or "papilliferous cystic carcinomas" when they contain papillae covered by irregular layers of cylindrical or polymorphous epithelium over a stalk composed of merely a blood vessel; these grow luxuriantly into the lumena and also into the interstitial tissue. The author saw such a case (fig. 902) in a 45 year old woman who had metastases in the axillary lymph nodes. (Compare Pusateri, papillary metastases.) See p. 1779, fig. 888, concerning the benign variety of these tumors, *i.e.*, the "cyst adenoma papilliferum," and compare with similar tumors of the ovaries. See p. 1785 concerning carcinosarcomas and carcinochondrosarcomas.

Etiology.—Occasionally, trauma is held responsible (blows, pressure); x-ray burns may initiate the process (see the case of Depenthal, bilateral breast carcinoma); influence of the sexual life (increased function?) is debatable; heredity, and according to the experiences of the author and others (see, *e.g.*, Verga), a previous chronic mastitis may be provocative. Carcinoma may also arise after a breast has been cystic for many years, or from adenomas, or adenofibromas, (compare the work of Kuru). In old solitary cysts there may arise a papillary adeno-carcinoma.

Course of Breast Carcinomas.—As a rule, only one breast is involved, and usually by one nodule; both breasts may, however, become the seat of two primary tumors, coincidently, or one after the other, (Silberberg, lit.); more frequently, however, one is a result of lymphogenous metastasis from the other. More rarely, the entire breast together with the skin suddenly becomes carcinomatous. Skin involvement hastens lymphatic transfer into the skin of the other breast, (according to Gerota in 65%–70% of the cases). The palpably hard tumor, at first painless, infiltrates the breast tissues, but in the beginning is movable under the skin and against the underlying tissues (pectoralis). The growth may be very slow, (years), or very rapid, particularly in pregnant women and nursing mothers, and new nodules develop, or the original tumor enlarges, often, although not always, painfully (lancinating pain); it becomes soft or remains hard, and adheres to the underlying tissues and to the skin, which becomes immovable over the tumor, and thinned. Occasionally, the tumor may shrink in toto, or in part, while it keeps on growing in the periphery and in the depths. Those showing a tendency to shrink are called "scirrhus." Rupture through the skin is particularly prone to occur in soft carcinomas; the skin is infiltrated, destroyed, and a carcinomatous ulcer appears with hard, board-like edges and a crateriform space covered with hemorrhagic debris.

In the neighborhood, many nodular or flat metastases, like a rosary, or a string of pearls, appear in the skin, although usually not at great distances (abdominal wall, back, and so on), (see Eitner and Reitmann).

Occasionally, small vesicles are filled with edematous fluid (see also Neustadt, Wegelin). Sometimes the skin in the region of the breast or its neighborhood, or even over an entire half of the thorax may be infiltrated; or, as in bilateral carcinoma, the soft parts of the entire thorax are transformed into a hard, board-like mass adherent to the underlying structures (cancer-en-cuirasse). The pleura is diffusely infiltrated. Microscopically, these cases usually prove to be of the round cell, small alveolar type. Occasionally also, nodular tumor masses grow through the skin like fungi and are covered with pus and dirty fluid. Retraction or fixation of the nipple is quite frequent. The tumor infiltrates the neighborhood of the nipple, shoves forward the surroundings, while the larger ducts are pulled inward (Konig); or the retraction or fixation of the nipple is caused by a retracting growth under the areola or under the nipple itself; the more subcutaneous fat, the more marked the retraction of the nipple. This retraction is not pathognomonic of carcinoma, however, but may be produced by chronic mastitis, particularly in fatty breasts, as well as by other tumors. Thus the author saw it occur in sarcomas and cystic adenomas. Perhaps a better sign of a small scirrhous carcinoma deep in the breast of a woman in middle age is a circumscribed wrinkling of the skin in a fatty breast (Morton).

The most ominous symptom in the course of a carcinoma is infiltration of the regional lymph nodes, usually on the same side, chiefly the axillary nodes which communicate with the subclavicular and cervical chains. Solid tumor strands may be found extending from the original tumor to these metastases, and tumor nodules may occupy the connective tissue between the nodes. Equally serious are infiltrations under and in the lymph channels in the pectoralis muscle, which are connected with the retrosternal chains. But infiltration may occur very early or late; the nodes on the opposite side are usually spared until late. They feel very hard. On the other hand, simple hyperplastic swellings of the axillary lymph nodes may be quite difficult to distinguish, even microscopically, from beginning carcinoma (p. 284), but as a rule, in simple hyperplasia, they are not as hard.

In examinations of carcinomatous axillary lymph nodes, tuberculosis is occasionally found. As a rule, the latter is secondary to tuberculosis of the lungs (Prym, Pickhan); but tuberculosis of the breast must not be excluded without second thought, even though it is fairly uncommon.

In the further stages, initiated early or late, and which lead to death in a few years, as a rule (exceptional case of Billroth, 20 years), there develops the cachexia of carcinoma. Distant metastases through the blood stream occur quite early in the soft forms, therefore usually in younger individuals, but the scirrhous forms are sometimes found also. In fact, as a general

rule, the course is always more rapid in younger individuals. In a 28 year old girl in whom there was no clinical suspicion, the author found a solid medullary carcinoma as large as a cherry in the right breast, with wide spread metastases in the entire skeleton with the exception of the skull (osteoplastic in parts), as well as in the lymph nodes of the neck, chest and abdomen. In a 25 year old woman with a clinically unnoticed scirrhous solid carcinoma of the breast as large as an almond, the author

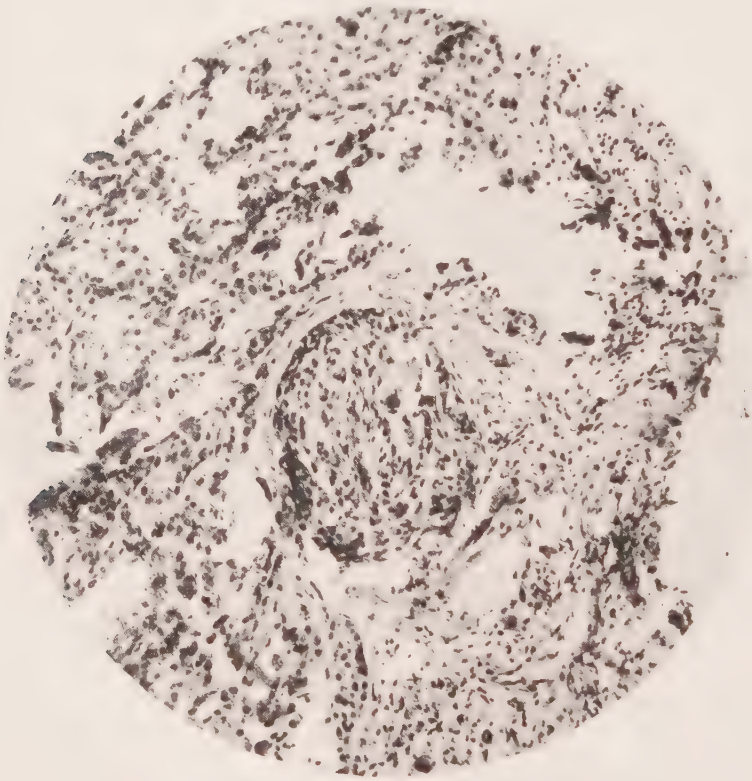


FIG. 903.—Thrombosis of a vein in the middle of a carcinoma of the breast. Such occurrences are quite common in the translator's experience, and many patients return after operation with dissemination throughout the body. Such "accidents" render fruitless many efforts at the grading of tumors. Doubtless, if many sections from a given tumor are examined, many tumors will show such pictures. × 150. L. H. #885.

found numerous metastases in the spinal column, sternum, ribs, pelvic bones (osteoplastic nature), in the liver, lungs, and pleural lymph vessels and in the adrenals. Since her skin was brown, the clinical diagnosis of Addison's disease was made; the duration after the beginning with pain in the back was only nine months. In older individuals, hard, scirrhous carcinomas are more frequent, and their course may be spread over years. Metastases occur in the pleura, liver, the bones (spontaneous fractures, see under osteoplastic carcinosis, fig. 642 p. 1224), and more rarely in

other organs, *e.g.*, the ovaries, wall of the stomach, uterine mucosa and so forth, and, as the author saw in a 53 year old woman 1½ years after amputation of the breast for carcinoma, in the chorioidea. (According to Bruns, in 71 cases of carcinoma in the bone which were accompanied by spontaneous fractures, the point of origin of the growth was the breast in 59.) After amputation of a carcinomatous breast, recurrence may take place quite early (according to the experience of the clinic of v. Bergmann, in over 40% it occurred in 6 months), in the scar or in the immediate neighborhood. It occasionally takes the form of a quickly spreading disseminated nodular recurrence which by confluence leads to a cancer-encuirasse reaching even to the crests of the ileii. Local recurrences may, however, appear quite late (the author saw the tumor recur 11 years after operation in a 52 year old woman, and in a 71 year old woman after 16 years). Recurrences are frequent in the regional lymph nodes and their neighborhood, by which the nerves and vessels, particularly the veins, are markedly infiltrated with the development of pain and edema of the arms. On the other hand, no local recurrence may take place and the scar may remain healed; or local recurrences do not take place but the other breast may be involved even some years after extirpation of the cancerous breast. If a tumor develop in the other breast years after one has been removed, thus, as in the author's case, in a 69 year old woman 14 years after amputation, it is probably better to assume that a second primary tumor arises. Metastases in the internal organs, and particularly in the skeleton, may appear after many years, thus, into the bones of the vertebral column when rupture into the spinal canal leads to compression of the spinal cord and paraplegia (fig. 979).

Gerota investigated the collateral and the retrograde course taken by the lymph after extirpation of a breast, and found that it passes toward the other breast, into the mediastinum and even downward into the inguinal region. This mode of spread was observed by the author in a case of inoperable carcinoma in a 70 year old woman; the tumor began in the right breast, spread to the left breast, occupied both axillae, infiltrated the cervical nodes, entered the mediastinum, and in the form of nodular strands spread under the breast and in and under the abdominal skin to the enlarged inguinal nodes. There were also many metastases in the bones.

According to Ritter, new lymph nodes may be developed in the fatty tissue and then become carcinomatous themselves. Preformed, constantly situated, lymph channels and stations are not previously present (see also de Groot, Ritter).

The combination of breast carcinoma with another primary carcinoma in some other situation is rare, thus, with a histologically different uterine carcinoma (Czerwenke). In a 54 year old woman, the author saw a

carcinoma of the urinary bladder demonstrable for four years, and a nodular carcinoma of the breast present for one year; the differing histological pictures excluded the latter as a metastasis of the former.

Cysts of the Breast. Parasites. Cysts.—On page 1764 we have discussed the so-called cystic mastitis and the so-called cystic breast, as well as the cystic epithelial and connective tissue tumors, but there still must be mentioned those cysts which appear as solitary structures or in small numbers. They may be as large as apples, filled with serous, often chocolate colored, contents, with a wall thickly covered with blood pigment and cholesterin, which may undergo calcification; they may be flattened, spherical or bulged. Such cysts are often due to trauma. Other solitary cysts may be filled with tumor masses (as in fig. 887). In many cases, the cysts arise from dilatation of a duct obstructed by a tumor. These may be termed "intracanalicular tumors." In other preformed cysts, usually varieties of adenofibromas, secondary tumors occasionally develop on their inner surfaces and fill the cyst more or less completely; the author has seen papillary fibroepitheliomas, papillary adenomas and papillary adenocarcinomas, as well as epithelial overgrowths with the character of a simplex or medullary carcinoma. Retention cysts present in groups in the ducts or on the surface of a breast, the seat of senile involution, are called involution cysts. These may be single or multiple, as large as peas, filled with serous, or colloid, green or brown pigmented, milky or caseous contents. In very small cysts, numerous psammoma bodies are sometimes found. These are in sclerotic connective tissue and come from retention following obstruction of the very smallest ducts. Large cysts may be due to retention or obstruction of the emptying of milk during lactation and are called "galactoceles." If the cyst ruptures, milk infiltration occurs. Very rarely, numerous squamous epithelial cysts contain cellular cholesterin debris; these are considered cholesteatomas (Konjetzny, lit.). Small cysts of this nature are not rare in fibroadenomas. Dermoid cysts of the breast are exceptionally rare. In a 45 year old woman, the author observed an epidermoid cyst as large as a hen's egg; it contained yellow, puriform, glistening masses, was situated in the depths of the breast and was changed into carcinoma; there was also present a coincident carcinoma simplex. Konjetzny described a papillary cystic cholesteatoma containing squamous epithelium which he found in the substance of the breast; there was carcinomatous change in part; he viewed it as a blastomatous teratoma, coming from embryonal ectoderm which still contained the potential ability to produce mesodermal structures of myxomatous and myxosarcomatous character. Bauer described an epidermoid cyst of the nipple. Small cysts lined with flat epithelial cells are probably retention cysts of the ducts.

Among the animal parasites, there might be mentioned the cysticercus (see Stumpf, in the deeper connective tissue near the pectoral muscle) and the echinococcus. Both are very rare. The author saw an echinococcus cyst as large as a hen's egg under the skin of the breast which gave the impression of a hard tumor (operative specimen). Literature concerning echinococcus may be found by Melchior. Borrel called attention to the relatively frequent acarus or demodex folliculorum in the ducts of the sebaceous glands around the nipple in mammary carcinoma, and he wondered if these parasites might not be intermediate hosts of the unknown poison producing the carcinoma. Orth (Tsunoda) found them, however, equally frequent in non-carcinomatous cases, and therefore disagrees with this idea. For the characteristics of parasitic lice, see Saul.

Among the vegetable parasites, may be mentioned thrush (see p. 560), which infects the areola and nipple of nursing women. The oidium albicans, otherwise an outspoken infector of mucous membranes, may establish itself in the folds of the skin of the breast as a thrush mycosis, and produce a lesion similar to eczema intertrigo (see Kumer, lit.).

CHAPTER X

NERVOUS SYSTEM

DISEASES OF THE MENINGES

Anatomy.—The tough, tendonous, glistening dura mater (pachymeninx) is also the periosteum of the internal surface of the skull. It is composed of two connective tissue layers rich in elastic fibers, the lamina externa and interna, the fibers of which cross almost at right angles. In children, usually until the seventh year, the dura is tightly attached to the bones. Normally, in adults it is loose on the convexities; at the base, however, particularly in the middle fossa, it remains fast. Adhesions along the convexity, as often found, particularly in older people, are pathological, and the result of an adhesive inflammation. Where the dura forms the venous sinus with its irregular lateral prolongations, the parasinoidal spaces, in which the veins of the diploe empty, it is divided into two leaves, and likewise where the nerves are embedded in it, thus, at the branches of the third to the sixth cranial nerves. Branches of the meningeal arteries course on the outer layer of the dura and are accompanied by sympathetic fibers (for more details of the vessels of the dura and the origin of the arterial sulci, see Demel). The efferent cranial nerves are surrounded by sheaths of dura; the sheath of the optic nerve reaches to the eyeball, those of the facial and acoustic nerves reach to the internal acoustic foramen.

The soft meninges, leptomeninges, are the arachnoidea and the pia. The arachnoidea constitutes the external membrane; toward the dura it is covered by a layer of endothelial cells and is avascular, while the pia, a plexus of vessels, held together by tender connective tissue, forms the inner meninx, lying directly on the brain (and the spinal cord), the convolutions of which it follows, and the vessels of which it surrounds with a sheath of connective tissue which is sent into the substance of the brain (and spinal cord). Between both meninges or leaves, there is a space, or a system of subarachnoid spaces, which contain the liquor cerebrospinalis; a network is spread from one meninx (arachnoidea) to the other (pia), the meshes of which are covered with endothelium. This meshwork binds the pia and arachnoidea into one whole (pia-arachnoidea, leptomeninx). In addition to the larger arterial and venous branches in this many chambered space, there are also the vessels of the brain which run

over the organ. The spaces are widest where the arachnoidea and the pia are separated over the greatest stretches, thus on the base, over the chiasm, and particularly at the transition place from the cerebellum to the medulla oblongata (cysterna magna cerebello-medullaris). The subarachnoid spaces communicate with (1) the subarachnoid space of the spinal cord, (2) the lymph tracts of the peripheral nerves (sheath of arachnoid along the optic and auditory nerves), (3) the ventricles medially through the foramen Magendii, (*i.e.*, the apertura mediana ventriculi IV at the posterior end of the fourth ventricle), as well as the two lateral apertures at the anterior end of the recessus laterales of the IV ventricle, where two stripe-like ends of the chorioid plexus are situated at the inner side of the flocculus (Key and Retzius), (4) through the intervention of the Pacchionian bodies with the sinus of the dura matris and the parasinoid spaces, (5) with the perivascular (periarterial, perivenous and pericapillary) lymph spaces of the brain. (But according to Held, see Spielmeyer, the true interadventitial lymph space, the Virchow-Robin's space, does not lie between the media and adventitia, but is represented, according to Schaffer, only by spaces in the glia which contain a varying amount of fluid.) It also communicates with the spaces which surround the ganglion cells. [For casts of the cerebral subarachnoid system, see Locke, C. E., Jr., and Naffziger, H. C., Arch. Neurol. & Psych. 12: 411 : 1924.—*Translator.*]

The arachnoid villi or Pacchionian bodies are granular, cauliflower-like outgrowths of the arachnoid, which push the thinned dura forward and jut into its venous sinus, or the parasinoid spaces. Cerebrospinal fluid may flow into the venous channels through the villi. These villi correspond to the Pacchionian furrows on the inner surface of the skull; they may even grow through the bone, and occasionally the translucent base of these furrows is pushed forward externally.

The pia penetrates the ventricle as the tela chorioidea which is a vascular membrane covered by true epithelium. The superior tela chorioidea enters through the oblique brain space and bridges over the third ventricle; from this there pass nodular vessel convolutions, the chorioid plexus laterales, through the foramen of Monroe into the lateral ventricles. The epithelium of the plexus and the ventricles secretes the cerebral spinal fluid. (Concerning the function of the plexus, see also the experimental results—vital staining with trypan blue—of Goldmann, and see in chapter on diseases of the ventricles.) The internal cerebral veins running in the superior tela chorioidea are collected at the posterior end of the pineal gland (epiphysis) to the unpaired vena cerebri magna (Galeni). The soft meninges are not tightly connected to the dura. Embryologically, all three meninges are composed of a connected mass between the anlage of the skull and the surface of the brain; later the subarachnoid

space is produced by separation of the parts (see Schaffer). The subdural space communicates with. (*a*) the deep lymph vessels and lymph nodes of the neck, (*b*) the lymph channels of the nasal cavities, (*c*) the subdural spaces of the nerve roots and the lymph channels of the peripheral nerves (optic, acoustic, etc.), (*d*) free lymph spaces in the dura; and (*e*) the venous sinus of the fura through the arachnoid villi. (For more details, see Hauptmann and v. Monakow.) See page 1843 concerning melanoblasts or chromatophores with brown pigment in the pia mater in the region of the medulla oblongata.

Diseases of the Dura Mater

Circulatory Disturbances and Inflammations.—Thrombosis of the sinus of the dura mater seldom occurs as an independent disease; it has few typical clinical symptoms; it often runs a latent course. There may be differentiated a bland (marantic) and an infectious thrombosis:

Marantic (primary, autochthonous) thrombosis appears particularly in the superior sagittal or longitudinal sinus and in the paired sinus transversus of small children and the aged, but also in poorly nourished young women, frequently in those who have had chlorosis. Sinus thrombosis may also follow the acute infectious diseases such as typhoid fever, etc. Hamburger believes the most important primary cause in sucklings is intestinal sepsis. At autopsy, the sinus is filled with dark red or brown coagula, which adhere to the wall and to the trabeculae stretched in the sinus. The communicating veins may also be filled with thrombosed blood. Occasionally, the cross section of a thrombus will show laminated layers. If filled with old, rough, brown, shrunken thrombi, the sinus becomes flat and shrunken. Recanalization takes place quite commonly, whereby the thrombus becomes cavernous (page 120).

Among the causes may be mentioned: cardiac decompensation, chlorotic, platelet-rich conditions of the blood (see page 189, under chlorosis), changes in the internal surface of the sinus, infectious or toxic (?) factors. According to Leichtenstern, cardiac weakness is the most important of the causes of thrombosis, but local conditions are no doubt of predisposing moment; thus sudden dilatation of the vessels with consequent slowing of the blood stream. The sinus is relatively stiff; it contains muscle; there is a network stretched across, in the meshes of which are situated the nodular Pacchionian granulations; thrombi are often seen on the latter. Perhaps fatty degeneration of the lining endothelium is also of importance (compare Thorel, Hanser, lit.). Vorpahl believes in the reverse course of events; in many cases hemorrhages into the brain occur first, and they are followed by thromboses. Such a possibility must be considered in individual cases.

The consequences of thrombosis, if it occurs rapidly and occupies a large part of the sinus, are stasis, edema, hemorrhages into the meninges and into the brain substance; the more of the sinus that is thrombosed, the more extensive are the hemorrhages; quite extensive red softening may appear in the brain.

The cerebral hemorrhages following sinus thromboses usually occur in the cortex. When the internal cerebral veins are thrombosed, however, there may be innumerable, thickly strewn, punctate hemorrhages, almost exclusively in the peripheral zone of the medullary substance or even in the central ganglia; it is exceptional to find large masses of blood (hemorrhagic infarction) as in figure 904.

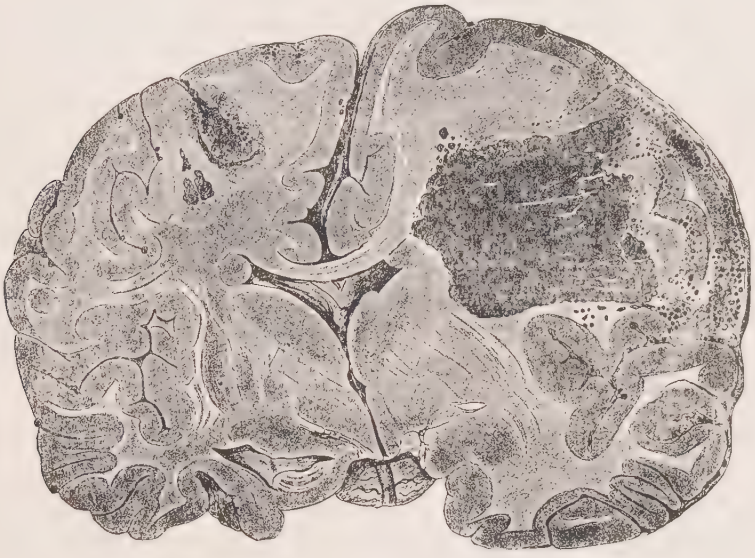


FIG. 904.—Large hemorrhage (stasis infarct) of white matter and many punctate cortical hemorrhages in sinus thrombosis; girl 1 year old; frontal section; 5/6 nat. size.

In an 18 year old chlorotic girl, the author found thrombosis of the posterior part of the longitudinal sinus, of both transverse sinuses, of the straight sinus, of the greater veins of Galeni, and of the chorioid plexus, with extensive red softening of the central ganglia, particularly the optic thalami. There was advanced fatty degeneration of the myocardium. Fever during the last few days of life. The author saw an analogous case in a 19 year old girl with chlorosis (narrow aorta and small, very flat, adrenals); fever toward the end to 41°, meningitic symptoms. In a 9 months old boy (diagnosis, meningitis), the longitudinal sinus, the transverse sinus, the straight sinus, the occipital sinus, and all of the veins emptying into the longitudinal sinus were thrombosed. There were pads of blood over the left hemisphere. Two-thirds of this hemisphere, exclusive of the basal ganglia, was softened and bloody; on section, thick

punctate hemorrhages, masses of coagula, and innumerable thrombosed vessels were found in the yellow, gray, and red softened cerebrum. More prominent cortical hemorrhages were seen by the author in a 16 year old and a 19 year old chlorotic girl, both of whom also died with the clinical symptoms of meningitis (sopor, stiff neck, and fever). In general, however, chlorotic sinus thrombosis is quite rare (Leichtenstern). A number of the author's observations have been published by Halff (in-Diss. Basel, 1904, lit.). Literature by Therman, who is of the opinion that local changes of infectious nature in the neighborhood of the sinuses are quite important; see also Bertier, Hawthorne, Herz.

Inflammatory (secondary) thrombosis, thrombophlebitis, occurs most frequently in one of the transverse sinuses in the sigmoid fossa in the mastoid portion of the temporal bone. At autopsy, the sinus is filled with discolored, purulent, or softened masses, the walls are yellow-green, decaying and easily torn; occasional abscesses may be seen in the neighborhood between the leaves of the dura (perisinus abscesses), or the dura is coincidently discolored and covered by a smeary or creamy pus.

The Causes of Inflammatory Sinus Thrombosis.—(*a*) Following otitis media with caries of the petrous portion of the temporal bone (see under suppurative meningitis), in which condition, the transverse sinus is the most frequently involved. This inflammation may spread (1) from the diseased bone through contact, or (2) from an extradural abscess in the wall of the sinus between the diseased bone and the sinus. Less commonly a thrombophlebitis (3) is spread directly or indirectly into the veins emptying into the sinus (small veins of the bones, the emissary mastoid veins, a dural vein, etc.). In unilateral otitis media the thrombophlebitis may spread retrograde through the torcular herophili into the transverse sinus and the bulb of the jugular vein of the healthy side; cases also occur in which spread of the thrombosis takes place from the jugular to the superior vena cava; in still other cases, the lungs are showered with infectious emboli, while the internal jugular on the diseased side is occluded by organization, and is obliterated to a mere string. (*b*) Non-otogenic causes. Thrombosis may be initiated in the sinus via the larger veins or small emissaries of Santorini coming from other bony parts, particularly from a periostitis of the maxillary bone, or following injuries to the skull, or deep inflammations of the external soft parts of the head, particularly the face and its cavities (mouth, nose, and sinuses, comp. p. 296). Thus a phlegmon of the orbit with panophthalmia may spread from the ophthalmic veins into the cavernous sinus, and lead to meningitis and pyemia; this occasionally occurs even from small furuncles, infected wounds, or erysipelas of the face. Phlebitis of the cavernous sinus has been observed in furuncles of the neck (Otto, lit.).

The author autopsied three cases of orbital phlegmons with bilateral panophthalmia, phlebitis of the ophthalmic veins and cavernous sinus, and suppurative, chiefly basal, meningitis, with pyemia. These occurred in younger individuals; the points of origin were a small 14 days old excoriation of the upper lip, a small comedo in the left part of the face, the individual having a phlegmon of the right hand, and a furuncle above the right eye; in all cases there were many abscesses in the lungs; staphylococci were found in vivo and post mortem in the blood and in the abscesses. In a 20 year old laborer with furuncles on the face (cheeks), phlebitis of the facial vein could be demonstrated spreading to the ophthalmic vein and followed by a phlegmon of the orbit, suppurative meningitis and pyemia. In a 16 year old girl with a carbuncle on the upper lip and a phlegmon of the right orbit, there arose an extensive edema of the lower eye lid but no meningitis or pyemia.

Consequences of Inflammatory Sinus Thromboses.— In addition to the mechanical consequences of stasis, the following conditions may be mentioned as resulting from the septic character of the thrombosis; diffuse suppurative leptomeningitis, extradural abscesses, and brain abscesses. In otitic sinus thrombosis, abscesses are particularly frequent in the cerebellum, in the sigmoid fossa of the transverse sulcus bordering the quadrangular and semilunar lobes. Pyemic metastases occur when loose thrombi are carried away, especially into the lungs, where they give rise to multiple or innumerable abscesses. The other signs of pyemia such as cloudy swelling and fatty degeneration of the parenchymatous organs, acute splenic tumor, etc., are, of course, also present. Bland emboli produce simple infarcts; such emboli might come from a diseased, but only partly septic, thrombosed sinus, or from the internal jugular vein.

Hemorrhages. Hematoma (Varix Sinus Sagittalis).— Small, usually multiple, hemorrhages into the tissues of the dura are found occasionally in asphyxia (dural hemorrhage). Hemorrhages between the skull and the dura (epidural hemorrhage, or extradural hematoma) come from torn vessels (arteries), *e.g.*, gun shot wounds or fractures of the skull; a blow on the head without fracture, particularly in older people with calcified, brittle arteries (middle and anterior meningeals) and deep meningeal sulci may lead to extradural hematoma. The epidural extravasation of blood found in people who have burned to death and in cremated bodies, appears, according to Strassmann, because the heat of the flames drives the blood from the diploe into the inner part of the skull (lit. by v. Horoszkiewicz and Leers); according to Reuter, they may also appear because the dura is lifted off by the heat and shrinks and thus forces blood from out the torn sinus. At autopsy, between the skull and the dura which is lifted from the bone, there is a tough, pressed out pad

of blood (cephalhematoma internum), which compresses the brain by its spherical or egg-shaped contour and thus smoothes out the convolutions. Horizontal sections best illustrate the condition. Clinically, there often appear paralyses or hemiplegias on the opposite side (contralateral). Occasionally, however, collateral hemiplegia occurs on the same side, and clinically gives rise to great difficulty in the diagnosis of the hemisphere compressed by the hematoma. The author has seen cases, in which trephining has been done on the wrong side. But this mistake has occasionally been made in actual lesions of the cerebrum itself (hemorrhages, abscesses, tumors, softening; see Ledderhose). Study of horizontal sections brings out the details more clearly (compare fig. 932; illustrations by Enderlen; see also Giesel, lit.).

Hemorrhages between the dura and arachnoid are called subdural hemorrhages or hematomas, or also intradural hematomas; the latter term is in general use, but is an incorrect designation; subdural hematoma is more accurate. In traumata the hemorrhages come from torn veins of the pia.

Of particular importance are injuries of the head during childbirth because they are followed by intracranial hemorrhages (cephalhematomas have been discussed on page 1092). There occur tears of the veins, particularly the free pial veins, which empty like bridges into the sagittal sinus; they are caused by displacement of the flat bones of the skull; occasionally the vena magna Galeni, which empties into the straight sinus, is torn, or even a transverse tear occurs of the transverse sinus in pelvic presentations. Tears of the tentorium (Beneke) take place through overstretching of the falx cerebri. These are usually in the upper leaf of the tentorium, which looks as though it were ruptured, or in the free thickened border of the tentorium, usually both leaves. The lateral pressure from temporal bone to temporal bone produces the overstretching of the falx through lengthening of the long axis of the skull and thus the tears are at right angles to these axes. The blood lies on the upper leaf, which is the continuation of the falx, or below the lower leaf, which becomes the dura of the cerebellum (peribulbar; according to Seitz, infra-tentorial hemorrhages). Large hemorrhages, particularly when peribulbar, may lead to death through brain pressure and asphyxia; in some cases with minor hemorrhage, death is probably due to lesions of the cerebrum itself. Many tears give no symptoms. Tears of the tentorium are very frequent, particularly during instrumental childbirth, version and extraction, but also from narrow pelves, spontaneous births through compression of a large skull, and even spontaneous too-rapid birth or premature birth. (See lit. by Pott, Bauereisen, Benthin, Sangemeister, Kowitz, A. L. Vischer, Beneke-Zausch, Schafer, Gabriel.) At autopsy, tears of the

tentorium are best demonstrated by the following method. The ordinary circular saw cut is carried to within about 1 cm. of the center, both anteriorly and posteriorly. Then the saw is carried from the anterior to posterior surfaces over the vertex about 1 cm. to either side of the midline. This leaves a bridge of bone running anteriorly and posteriorly about 2 cm. wide; the falx and the longitudinal sinus lie under this bridge. Through the windows on both sides the hemispheres of the cerebrum are carefully lifted out one after the other, and are separated above the bridge through the crus cerebri. Then the falx and the tentorium can be examined. An easier and quicker method is to section the brain horizontally through the ordinary horizontal opening of the skull. The larger mass of the cerebrum is lifted off with the roof of the skull. The remaining parts of the cerebrum in the basal part of the skull may easily be lifted out, and after separation of the base of the brain, the tentorium can be observed.

Another cause of larger subdural hemorrhages during birth arises from oblique pull and tearing of the intervertebral capsules of the joints of the cervical vertebral column (Stoltzenberg). These hemorrhages into the vetebral canal and the meninges may reach the posterior fossa of the skull and cause death. Schultze's method of artificial respiration has also been held responsible; of course, it can only produce further harm in already existing tentorium tears (see M. Hirsch).

See v. Kruska for injuries of the brain substance by traumatic hemorrhages intra partum, and their consequences in after life such as atrophies, scleroses, cysts, porencephalia, and their clinical pictures as well as cerebral paralyses of children, epilepsy, etc. Edinger believes these are due to sclerosis of the hippocampus major as consequences of birth injuries, especially tears of the tentorium.

The spaces containing blood situated over the bones and under the soft parts of the skull, are called the sinus pericranii (Stromeyer); they are said to communicate with the sinuses of the inside of the skull through spaces in the bone. One group must be differentiated as a varix herniosus sinus sagittalis (Heineke) which may be presented as a cephalocele (lit. by A. Muller, Morig); this is of the nature of a developmental failure.

Pachymeningitis Interna Hemorrhagica.—In this condition, there are found on the inner surface of the dura, particularly along its convexity, and often only unilateral, more or less transparent, easily removable membranes stained diffusely, or spotted red, dark brown or rust brown by blood pigment. They lead to considerable thickening of the dura.

The classical picture of this condition which is a clinically important, spontaneous, progressive disease, is seen chiefly in men of advanced or old age, in many psychoses and atrophies of the brain, occasionally in

paralytic dementia, in heart and kidney diseases, arteriosclerosis, and particularly in chronic drunkards.

On the other hand, minor pachymeningitic hemorrhagic membranes are frequent in infectious diseases both in adults with sepsis, typhoid fever, etc., as well as in children with scarlet fever, whooping cough, (Hada, lit., Roth), congenital syphilis (Schmincke), focal softening of the surface of the brain, gummatous or tuberculous or carcinomatous infiltrations of the dura, tumors of the dura, etc.

Spotted, bloody, skin-like deposits are also seen occasionally in the hemorrhagic diatheses such as pernicious anemia, leukemia, carcinoma and others.

Traumatic cases should be differentiated from ordinary hemorrhagic pachymeningitis, because in otherwise healthy people, traumata and aseptic hemorrhages are not sufficient to produce progressive pachymeningitis (Boeckmann, Wolff); the hemorrhages are organized to connective tissue membranes. Exceptionally, the condition seems to follow "birth traumata," and Schmincke has described such a case following forceps extraction which also showed multiple scars in the glia of the cortex; these childhood cases, especially those of sucklings, are often initiated by marked serous exudation (lit. by Wohlwill, Rosenberg, Schmincke); preference for the convexity is, however, not so prominent (Doehle).

On the basal dura there are occasional isolated, pale, fibrinous, cobweb-like membranes, poor in leucocytes and easily washed off. This is the beginning of the process, after which cells and vessels grow from the vascular layer of the dura into the fibrin as a granulation tissue. From this, a tender connective tissue develops, in which the chief cells are spindle-shaped, and in its meshes there is a rich, yellow pigment. This pigment appears also in the endothelial cells, the round cells, and free in the tissues. The connective tissue membrane contains many wide, thin-walled vessels coming from the dura; these are often convoluted and varicose, and show tendencies to hemorrhage, particularly when congested. The hemorrhages are usually minor, but they leave red and brown residues of blood pigment, and irritate new membrane formation. Thus new membranes arise from continuous absorption of the blood and renewed hemorrhages, while the older tissues become thickened, poor in vessels and infiltrated by pigment, which is usually granular, and contained in oval or spindle-shaped cells; occasionally calcium bodies, the so-called psammoma bodies, are also present.

In this presentation, we have followed the description of Virchow, which was later confirmed by Melnikow-Raswedenkow, viz., that an inflammation particularly rich in fibrinous exudate, similar to that occurring in other serous membranes, is the primary condition. Melnikow

believes that toxic damage to the dura is the cause of the inflammation. Others, particularly Huguenin, consider hemorrhage from diseased and abnormally permeable vessels as the primary cause, and the membrane formation as merely an organization of the coagula. In the cases occurring in the hemorrhagic diatheses, the causative relationship of the hemorrhage must probably be recognized. Perhaps, however, the cause is an, as yet, unknown toxin (?) (see Wohlwill). Jores and his pupils, (van Vleuten, Laurent), believe that the condition starts as an hyperplasia of the subendothelial capillary layer of the dura, which produces very

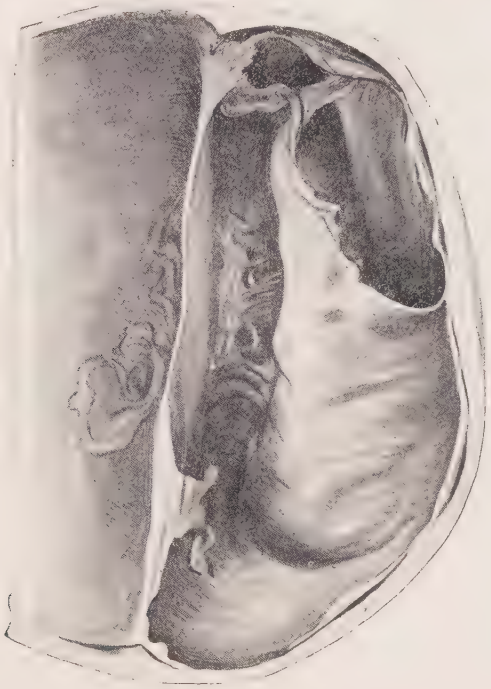


FIG. 905.—Older hematoma of dura mater; immense, pigmented, brawny, partly calcified sac containing blood; left cerebral hemisphere showed a deep depression corresponding to this; ½ nat. size.

vascular but poorly cellular membranes; the hemorrhages are secondary, and are organized in the ordinary way to poorly vascular, richly cellular connective tissue membranes. This viewpoint has lately been adopted by Wohlwill for the great majority of cases; it is said that a damage to the endothelial cells initiates the process. The external side of the dura, as far as there are no traumatic hemorrhages, is rarely involved by the disease. But the author saw a case in which the organized tissue looked like a large thin rag with practically no connections with the dura, upon which it lay and from which it could be lifted after the top of the skull had been removed.

If a larger hemorrhage separates the membranes, there arises a so-called hematoma of the dura mater, which is often egg-shaped, reaching the size of a thumb or even a fist; it is covered by dura (intra- or subdural), is of moderately soft consistency, lying over the inside like a pillow, or covering the brain like a cap and compressing its structures.

Apoplectic attacks may occur with loss of consciousness and unilateral paralysis, and death may follow the increased intra-cranial pressure. The conditions can be well demonstrated at autopsy by making horizontal sections of the brain in situ.

FIG. 906.—Large hygroma of dura mater; sac contained watery fluid.

Even large hemorrhages into the membranes may be absorbed leaving behind circumscribed, perhaps many-chambered cysts with more or less thick, sometimes scarred or calcified or ossified, walls, and a clear white or yellow serous content (hygroma durae matris, Virchow).

Occasionally the hygroma may be a cap-like bladder covering the entire convexity of one hemisphere; it may be loosely connected with the inner surface of the dura and lie between the brain and the dura; see the author's case, fig. 906, and similar case described by Potoschnig, lit.

Blood may still be contained in very old sacs, (fig. 905).

O. Hildebrand believes it possible that these hygromas may also arise from old hemorrhages which separate the dura, as well as from lymph cysts (?), in addition to the method as outlined by Virchow.

External pachymeningitis involves the external periosteal layer. It is usually suppurative or gangrenous, does not arise spontaneously but through spread from the neighborhood, *e.g.*, in erysipelas, infectious wounds,

tumors or caries of the skull, particularly caries of the petrous parts of the temporal bone, or suppurative sinus thrombosis. The dura is covered by pus, and is dark green or gray yellow. Much pus, (extradural abscess), lifts the dura from the bone. The greatest danger lies in spread to the sinus. The inner surface of the dura may remain intact, or the inflammation spread (meningitis), or a brain abscess develop.

After trauma without fracture, *e.g.*, a blow on the head, or in older people, the dura is occasionally found adherent and thickened (chronic adhesive pachymeningitis).

Pachymeningitis chronica fibrosa may lead to diffuse, leather-like thickening of the dura. It becomes focally adherent to the skull, the inner table of which may be thickened and cloudy. Not rarely, there are adhesions to the other membranes also.

In rare cases, a fetal chronic exudative internal pachymeningitis hinders the development of the brain (Rubesch, lit.).

Infectious Granulation Tumors. Tuberculosis of the Dura.—Miliary tubercles, less commonly, diffuse productive tuberculous inflammation, very frequently follow acute tuberculosis of the pia-arachnoidea; indeed, according to Ribbert and Chiari, it is a constant accompaniment. The tubercles are often very small and hardly visible. They are found in largest number at the base of the brain, particularly over the alae minores of the sphenoid bone. Such a tuberculous pachymeningitis arises from contact infection from the leptomeninx. Confluent caseous tubercles are seen in tuberculous caries. Larger conglomerate tubercles which occasionally appear in the basal dura in the region of the clivus, may be mistaken for tumors (ecchondroses).

Syphilis is found most frequently as a gummatous pachymeningitis following syphilitic caries of the bones of the skull, although it may be established primarily as a gummatous pachymeningitis, which involves the bones secondarily. There appear distinct flat gummata in an otherwise normal dura, or, more uncommonly, diffuse, pale gray red or caseous thickenings of the entire dura, which is covered by a grayish yellow gummatous layer, even as thick as one centimeter. The condition may easily be mistaken for primary tumor, or a metastatic carcinoma! Adhesions to the soft membranes are very frequent. In constitutional syphilis there may also appear a fibrous thickening of the dura (pachymeningitis fibrosa), in which the skull itself is diffusely thickened.

Actinomycosis of the dura arises from actinomycotic encephalitis, as well as from actinomycotic processes in the basal parts of the skull.

Leukemic infiltrates appear as diffuse growths or as flat plates on the inner surface of the dura, or externally corresponding to a periosteal infiltration.

Tumors of the Dura Mater. Primary Tumors.— The tumors arising from the dura belong to the group of connective tissue tumors. A number are histologically, and in relation to their surroundings also, benign; these are mostly circumscribed fibro-endothelial tumors, often calcified and then called psammomas. Others, in spite of benign histological characters, are dangerous because of pressure on the surroundings, or because they burrow deep into the cerebrum (fig. 910). They never enter the lymph channels, however, and are always separated from the brain substance by connective tissue (Kron). Others, which are also closely related to the fibro-endothelial tumors are, however, so richly cellular that they must be considered sarcomas; furthermore, they invade the neighborhood in their growth; other sarcomas, *e.g.*, the round cell, have no histological relationship to the endothelial tumors.

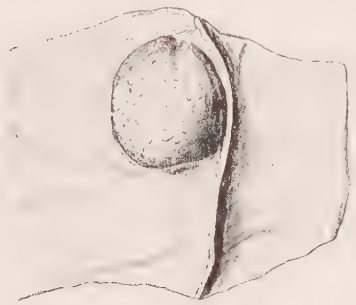


Fig. 907.—So-called psammoma of dura near the falx; nat. size.

The **fibro-endothelial tumors** of the dura are most frequently circumscribed benign growths which have been called by Virchow, "psammoma," a poor term. They appear as hemispherical, broad based, simple, or primary multiple excrescences, seldom larger than cherries or small apples. They grow into, and compress, the neighboring brain substance, to which, as a rule, they are loosely attached. They are tightly adherent to the dura, however, but some may be easily plucked off and are attached loosely through vessels and strands of connective tissue. Most of them are quite hard; not rarely they are sandy to the touch, red gray yellow or yellow white in color and composed of lobules and granules so that they imitate a mulberry. Their pliability in general is quite variable; some are dense, fleshy; others are alveolar or so soft that the sand granules fall out; others are composed of a loose meshwork. The sites of election are in the neighborhood of the sphenoid bone and the falx, particularly the anterior part of the latter (fig. 907).

They are most frequent in old age, but Sternberg has described a case occurring in a 19 year old individual.

Histologically, they vary, but their basic characteristics are made up of a vascular connective tissue in which are embedded flat or thick endothelial cells. These are similar in part to those which are isolated or are embedded as villi in the dura and are found on its surface. But many types of structure may appear through regression of the endothelial parts, whereby the tumor becomes a poorly cellular fibro-endothelioma, or even a fibroma, while in other cases the endothelial cells are predominant and

the tumor is richly cellular and similar to a sarcoma; in still others the fibrous and endothelial parts are about in equal masses and there thus appear fibro-endotheliomas. All stages of transition from poorly cellular to very richly cellular types occur. Further variations are brought about by differences in the arrangement of the connective tissue and vessels; in one tumor the connective tissue is composed of long fibers, enclosing flatter or thicker endothelial cell masses (fascicular, plexiform fibro-endothelioma); in other tumors, meshes are formed which contain endothelial cells (alveolar fibro-endothelioma). The more numerous the cell masses, the more marked is the sarcomatous character (alveolar sarcoma, endothelial sarcoma). A further variation is introduced in the fibro-vascular part by the richness of blood vessels which surround the cell

FIG. 908.

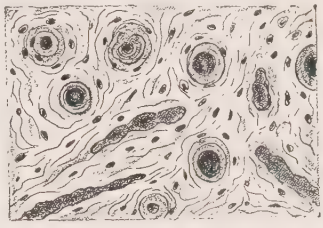


FIG. 909.

FIG. 908.—Calcium granules from a psammo-fibroma of choroid plexus; mostly cross sections of these bodies; in two places a mulberry-like configuration. (High power.)

FIG. 909.—From a poorly cellular psammomous fibroendothelioma of dura mater of cerebrum. Fibrous ground work tissue containing hyalin globules partly nucleated and derived from hyalin endothelial cells which enclose calcium bodies with concentric laminated structure; calcium spicules formed by petrifaction of hyalin connective tissue. (High power.)

masses concentrically or in longitudinal directions; if the cell masses are few in number, the tumor looks like a hemangioma; if the cell masses are numerous, the picture of perivascular angiosarcoma is reproduced. If the vessel walls are hyalin and thickened—a frequent finding—another type of picture is presented (see under spinal cord). The variety of pictures explains the varied opinions concerning these tumors (Lindner). We will mention still a few other particular forms. M. B. Schmidt arrives at the opinion that these tumors are of arachnoid origin, coming from the endothelial cells, which, physiologically analogous to the Pacchionian bodies, are pushed, into the dura as independent villi, cell masses, etc., from the surface of the arachnoid (see Aoyagi and Kyono); they are covered by the innermost layer of the dura. Thus the tumor assumes the shape of an hour glass as it grows through the dura and is covered by a capsule from the inner dural layers, (see, among others, Antoni). On the other hand, Ribbert believes that the endothelial cells come from the surface cells of the dura, and he, as well as Prym, disagrees with Ficks who, until now, believed that the tumors which are considered endotheliomas,

are, in reality, epithelial. Ricker believes that an epithelial genesis is quite possible.

Translator's Remarks.—The work of Mallory and of Cushing places the origin of these "meningiomas" in the arachnoid villi. The cells are fibroblastic in nature. See Mallory, F. B., J. Med. Res., 41: 349: 1920, and Cushing, H., Brain, 45: 282: 1922.

It is also possible that they have some sort of a characteristic peculiarity, inasmuch as the formation of the psammoma is so frequent.

FIG. 910.—Large, very vascular psammomous fibroendothelioma of dura (D), not malignant histologically, but prolongations in the brain. The apparently isolated smaller round nodules in the frontal section are attached to the main tumor. Sharp demarkation of tumor nodules against the displaced—not infiltrated—brain substance. P, prolapse of brain alongside the tumor, developed after trephining; B, corpus callosum; Ci, internal capsule; above this the corpus striatum; below the lenticular nucleus; externally the claustrum is visible; Oe, oculomotor nerve, internal, the optic nerves; s, temporal lobe; from a man 25 years of age; ¾ nat. size.

Psammoma bodies (corpora arenacea) are sandy inclusions, also found independent of tumor formations as concentrically layered calcium-containing bodies, (brain sand), in the dura mater and the choroid plexus under normal conditions as well as in chronic inflammatory processes (p. 1812) (fig. 908). They arise by calcium deposits in a hyalin ground work (Ernst), *i.e.*, in hyalin connective tissue fibers, in hyalin vessels, and

in concentrically layered hyalin endothelial cell masses. In other situations, *e.g.*, ovarian tumors, they come from hyalin cell products. They begin as rounded laminated concretions or pointed, cactus-like or trabeculated nodular petrefactions, which become laminated through the successive deposit of new hyalin and calcified masses. In the brain substance there occur psammogliomas; in other situations, *e.g.*, the ovaries, psammomous cyst adenomas and cystocarcinomas (see pages 1562 and 1573). These must be differentiated from acervulus in the narrower sense; these latter are the sandy bodies in the pineal gland, which are parallel striped like mulberries, and undergo calcification to the surface; after solution of their carbonic acid they allow recognition of a layered albuminous material of unknown origin, just like true stones. (For more details, see M. B. Schmidt.)

Sarcomas arise from the external periosteal, or the internal layers of the dura. In the former event, they rupture through the bones and spread from the cranial cavity into neighboring cavities. They are then lobulated or polypoid and endanger the neighboring cerebral parts or invade the brain itself. They may reach the size of fists or larger, prefer the base, and often produce severe symptoms, inasmuch as they compress and compromise various parts, or invade them.

See Rossle, concerning an osteosarcoma of the dura of the frontal bone having a possible traumatic cause after a gun shot wound—2½ years elapsed between the wound and extirpation of the tumor.

Histologically, the most frequent forms are fibrosarcomas and spindle cell sarcomas, but among them are also found bone-forming osteosarcomas, and, less often, round cell, and polymorphocellular sarcomas. Many sarcomas are alveolar in arrangement; others are hemangiosarcomas (lit. Corten); others are telangiectatic. The greater the length of the cells, the harder the tumor. If the interstitial substance is mucoid there appear myxosarcomas. Psammoma sarcomas occur through the formation of psammoma bodies in sarcomas of the dura; they are particularly frequent in spindle cell sarcomas. These latter have a tendency to be arranged in concentric layers, which point to a close relationship, and perhaps even a general origin with the benign endotheliomas; in fact, this is true in general of sarcomas of the dura, which become endothelial sarcomas only through the fact that they are richer in cells than endotheliomas.

Rare forms of sarcomas have been pointed out elsewhere, (thus on pages 178, 1211, 1212, and see under spinal cord). Among the rare forms of dural tumors may be mentioned the following: in many "psammomas" there is recognized a fibrosarcomatous background containing numerous blood vessels, the endothelial cells of which are markedly overgrown. They may form endothelial pearls (oblique section), which often become

psammoma bodies, while in other situations long endothelial villi jut into the vessels, become hyalinized, and finally calcified (fig. 911). The name usually given to this tumor is sarcoma angiolithique (Cornil and Ranvier).

If masses of cells are predominant, and if, in them, there occur only naked vessels, *i.e.*, those not surrounded by connective tissue, with hyalin walls, and if the cell masses produce collars around the vessels, as particularly found in the borders of the tumor, the name given may be angiosarcoma, or perivascular sarcoma, with hyalin degeneration of the vessel walls (sarcoma cylindromatosum).

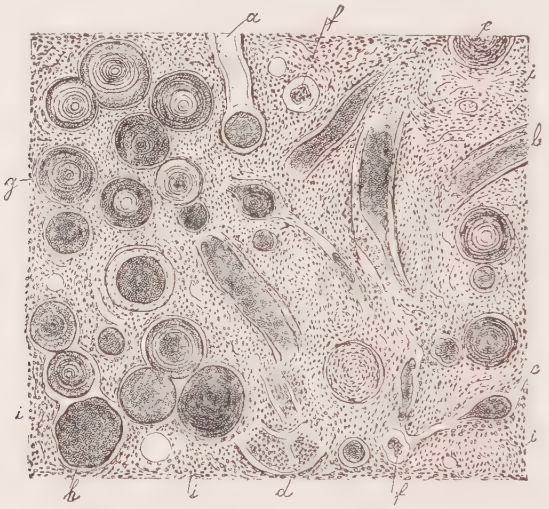


FIG. 911.—Psammo-sarcoma of dura mater. Fibrosarcomatous ground tissue (l) within numerous laminated (g), non-laminated (h), rounded psammoma granules, and calcified masses. The endothelium of blood vessels proliferated (b); in places the lumena filled by cells (d, e, f) or stratified or non-stratified, partly calcareous masses, cut lengthwise, oblique or across (b, a); at f, vessels with hyalin walls, the lumena filled with cells; vessel a, contains blood. (Low power.)

But hyalin change of the vessel walls may be lacking, and the cells of the adventitia may proliferate and produce epithelial-like cells (perithelium) around the capillaries. The cells may also radiate from the vessel walls such as is hinted in figure 632, which represents a type of perivascular sarcoma.

Smaller or larger meshes of vascular connective tissue surround masses of cells which are either flat and like endothelial cells, or thicker, and rounded or angular, like epithelial cells. In the tumors with flat cells, the latter may become concentrically layered, or the cell masses are turned as a whole in their long axes, and on cross section appear as whorled figures. Central necrosis often takes place in larger cell masses. This form has been called alveolar endothelioma; but the name large cell alveolar sarcoma may also be used (fig. 631, p. 1211).

Fibromas of the dura are met usually as soft vascular psammomous types.

Osteomas of the dura occur as osteophytes on the outer surface, or as long plaques of bone, or as pointed nodules in the tissue of the thickened,

FIG. 912.—Rare form of psammo-sarcoma of cerebral dura mater. Hemangiosarcoma, *i.e.*, perivascular sarcoma (perithelial sarcoma) in which capillary blood vessels (a-1, a-2), with their endothelium visible are surrounded by cell envelopes, some vessels contain blood (a-1); sectioned endothelial nuclei apparently lie in the lumen. The sarcoma cells are well preserved (b-1), or have undergone vesicular and hyalin change (b-2), or are metamorphosed into a hyalin mass (b-3) without nuclei. Nuclear figures vary and are often degenerated. At c, calcification (intensive blue stain) of degenerated cells. Hematoxylin stain (high power).

or normal, or even thinned dura. In the falx they often appear like plowshares (fig. 913).

FIG. 913.—Osteoma of falx cerebri; situated sagittally between frontal lobes; from an older man (suicide by opium); nat. size.

Calcification of the pachymeninx, occasionally in the form of yellow white, macroscopically visible, stripes and spots, occurs in marked senile osteoporosis of the vault of the skull as well as in usura cranii (brain pressure in brain tumors; see Chiari, Nunokawa, Allenbach, Saltykow). There is local transport of calcium, particularly in the outer layers of the

dura. Minor (microscopic) degrees of calcification occur from the sixteenth year of life onward, as a physiological process. From the fortieth year of life onward, the dura, which then is tightly adherent to the skull, is often markedly calcified (H. R. Schmidt).

Metastatic Tumors. Carcinomata and sarcomata have been observed; both may occasionally produce diffuse thickening of the dura and adhesions between this membrane and the bone, or in the form of nodules, usually on the inner surface. Occasionally, carcinomata take the form of soft rounded plaques, and the bones are flattened or infiltrated where they are situated. In melanosarcomatosis the dura may appear as though rubbed

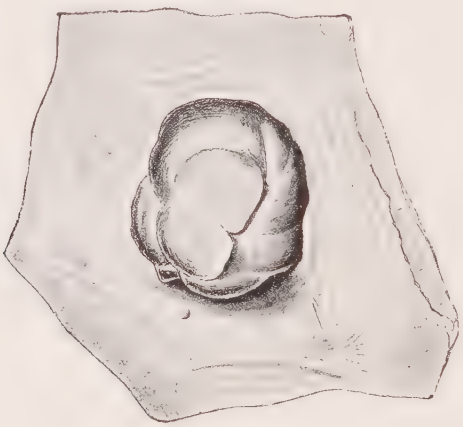


FIG. 914.—Metastatic sarcoma nodule the size of a walnut in dura from an angiosarcoma of retrobulbar tissue; girl 25 years old.

with ashes. It is not always easy to determine whether the metastasis belongs to the bone or the dura. Secondary sarcomas of the bones of the skull, or the brain, etc., occasionally infiltrate the dura.

Diseases of the Pia-arachnoidea

Circulatory Disturbances. Anemia and Hyperemia.—These occur only in the pia, since the arachnoid contains no vessels. Anemia is brought about by general anemia, or by local causes, *e.g.*, an increase of intracranial pressure in internal hydrocephalus, tumor of the brain, etc.

Hyperemia.—(In the differentiation of veins and arteries on the brain surface it will be noted that the veins lie on the surface, and the larger arteries in the depths of sulci.) Active hyperemia or congestion is difficult to determine in the cadaver. Usually the hyperemia found at autopsy is due to stasis.

The amount of blood normally present in the brain of the cadaver varies considerably; a difference is apparent if the head is opened before or after the remainder of the body; in the latter case, the jugulars, the sin-

uses, and veins of the cerebral surface are emptied previously. In sudden death, there may be marked filling of the cerebral vessels. When an individual dies while lying on his back and remains so for a long time, the posterior vessels are markedly filled (post mortem hypostasis).

In all cases of asphyxia, *e.g.*, obstruction to the trachea, there is a stasis hyperemia in the veins of the pia. The veins of the surface are markedly filled, and the entire pia is as though injected to the finest branches, which appear blue red to blue black.

In stasis of longer duration, there is thickening of the soft meninges (see under chronic leptomeningitis, p. 1831). For causes of stasis, see under stasis in the brain, p. 1880.

Edema of the soft meninges, hydrops meningeus or hydrocephalus externus, is an accumulation of fluid in the subarachnoid spaces. As the edema becomes more marked, the sulci are stretched, and the soft meninges, which form a thick edematous layer particularly in the dependent parts, may be easily removed from the surface of the brain.

Exceptionally, marked edema may occur in the newborn after long, continuous dependency of the head in protracted labor.

Edema may occur as a complementary condition in atrophy of the brain, hydrocephalus externus ex vacuo; in stasis, general, as well as local; in hydremia, *e.g.*, in nephritis; as an inflammatory condition accompanying acute inflammations of the meninges and the substance of the cerebrum (compare under inflammations). See Stillman concerning edema of the pia-arachnoid.

Hemorrhages into the subarachnoid mesh-work are called subarachnoid hemorrhages. Bleedings on the surface of the arachnoid in the subdural space are called subdural. Hemorrhages into the brain substance which extend under the pia are called subpial; they frequently extend also into the subarachnoid space. Small hemorrhages into the pial tissue are called pial.

Subarachnoid hemorrhages occur in some cases from stasis, *e.g.*, following sinus thrombosis, or, more rarely, in cardiac and lung diseases. The most frequent causes are traumata of varying kinds, in which it must be remembered that the larger arterial and venous cerebral vessels lie in the subarachnoid space.

They may occur from difficult passage of the head during labor, as well as through application of the forceps to the skull, particularly to the parietal bones, which are forced over one another; the veins are torn; thus are explained a number of cases of sudden or apparent death of healthy newborn children, in which, however, subdural hemorrhages occur, (Kundrat, Kowitz). As further causes may be mentioned: fracture of the skull, or blows, or falls without fractures, cerebral hemorrhages in tumors, or ordinary apoplexies which rupture through the cortex, rupture

of a basal aneurysm. If a hemorrhage takes place into the lateral ventricles, the blood may follow the plexus into the subarachnoid spaces and collect in larger amounts above the medulla, the pons and bordering parts (fig. 933). Subpial and pial hemorrhages occur in various blood diseases, such as scurvy, purpura, leukemia, pernicious anemia, phosphorus poisoning, icterus gravis, hemorrhagic diatheses, tumors and bacteremias, as well as in influenza septicemia. Hemorrhagic leptomeningitis, perhaps with hemorrhages into the cerebral substance (leptomeningoencephalitis acuta), also occurs in anthrax. The lymph channels are no doubt the most frequent routes of spread from a focus in the face or in the neck (Fulci, lit., Herzog). The author has published a case of leptomeningitis anthracica in a case of inhalation anthrax (lit. by Risel).

Inflammations of the Pia-arachnoidea (Leptomeningitis) or Meningitis

Serous Leptomeningitis or Inflammatory Edema of the Pia-arachnoidea.—The inflammation may be general or localized. The subarachnoid space and the tissues of the pia are saturated with edematous fluid. As the dura is lifted off in general edema, both hemispheres, particularly their middle and lateral parts, are covered by serous, slightly cloudy fluid containing leucocytes and, less frequently, lymphocytes. The dependent parts are, as a rule, more edematous. The base shows minor involvement.

The fluid is contained in subarachnoid meshes and in the pia, and cannot be washed off, but may be displaced. The pia is the more strongly injected. The pia-arachnoidea is brittle. The ventricles usually contain an increased amount of cloudy fluid (acute internal hydrocephalus); the chorioid plexus is swollen; the brain is heavy.

Acute serous meningitis (Quincke, Boenninghaus, Wexel, Beck, Grabs, lit.) occurs as an initial stage, or as an accompanying condition in other, particularly suppurative, acute meningeal inflammations following otitic processes, diseases of the accessory sinuses of the nose, suppurative inflammation of the bones of the skull, or as an independent entity. In sun strokes, which are quickly fatal, a serous meningitis may be found; it may furthermore be found in general infectious diseases such as scarlet fever, measles, articular rheumatism, whooping cough, pneumonia, influenza; death from serous meningitis follows in a few days; the same condition is found occasionally in typhoid fever (Eichhorst, Loeb, see p. 795), and the corresponding bacteria may be found in the suppurative forms. Youthful individuals seem preferred in this, as well as in the chronic forms. The course, in contrast to suppurative or tuberculous meningitis, is usually favorable, but serous meningitis may produce severe symptoms of increased intracranial pressure and blindness (atrophy of the papilla). After simple exploratory operations, recovery often takes place, although

occasional recurrences are seen (lit. by Muskens). Concerning pretended relationship to the abnormally high skull, see p. 1190. In otitis media, rapid fatality occasionally occurs due to streptococcus meningitis, in which there is no pus discovered, but merely congestion and edema, *i.e.*, the inflammation is halted on the road to suppuration (Hutinel, Muck).

In chronic serous meningitis circumscripta (cystica), there appear local collections of fluid with cyst-like boundaries which follow obstruction to the outflow of fluid in the membranes through adhesions in the arachnoideal meshes (Krause and Placzek, Raymond and Claude, Axhausen, Muskens and Snellen, Oppenheim and Borchardt). Not only are the communications with the opened subarachnoid spaces closed, but even an outflow into the perivascular lymph spaces of the brain is rendered impossible. If the accumulated fluid is increased by transudation from over-permeable blood vessel walls, the "cyst" quickly, or slowly increases in size, and with it, the tension. It then exerts a pressure on the brain (or spinal cord), just as a brain tumor, with the symptoms of irritation, loss of consciousness, neuritis optica, etc. The change may be primary, a lepto-meningitis, the etiology of which, in general, is similar to that of diffuse serous meningitis, even though, according to Claude (lit.), syphilis and tuberculosis are important factors; or secondary as an accompanying change of cerebral or spinal diseases, particularly traumata of the skull or the spinal column (contusions, gun shot wounds) (Payr, Bittorf, traumatic serous meningitis). The condition usually occurs in the neighborhood of the cerebellum and the upper part of the spinal cord (Schlesinger, Schuster), more rarely in the cerebrum (Wendel, lit., Schultheiss, lit.). These "cysts" may later become abscesses (Kuttner).

Suppurative Meningitis.—By this term are understood inflammations of pure suppurative or sero-purulent or fibrino-purulent character. The exudate lies chiefly in the subarachnoid space and in the tissues of the pia, although some may lie outside of the arachnoidea. The soft meninges are infiltrated by a cloudy yellow white thick, or green yellow purulent exudate. Masses of pus frequently appear as stripes along the pial veins, or as collars around the veins in the subarachnoid space, or as spots or diffuse discolored stripes, so that the veins are more visible; it also collects in the spaces and folds where the subarachnoid space is widest, thus at the base, about the cysterna chiasmatis, and around the cerebello-medullary angles. Over the frontal and parietal lobes, it may form a thick pillow, which hides the markings of the brain surface (the so-called green hood, particularly frequent in epidemic cerebrospinal meningitis). The cortex may be saturated with serum. Occasionally there are hemorrhages, either in the meninges alone or coincidently in the cortex. The brain as a rule is voluminous and heavy.

An encephalitis of deeper parts, particularly the basal ganglia, may occur through extension of the infection along the perivascular lymph spaces (Sparbrod).

Usually there is a coincident acute internal hydrocephalus consisting of serous or purulent exudate. The former is merely because of obstruction to the communications between the ventricles and subarachnoid space; the latter is an extension of the inflammation along the plexus into

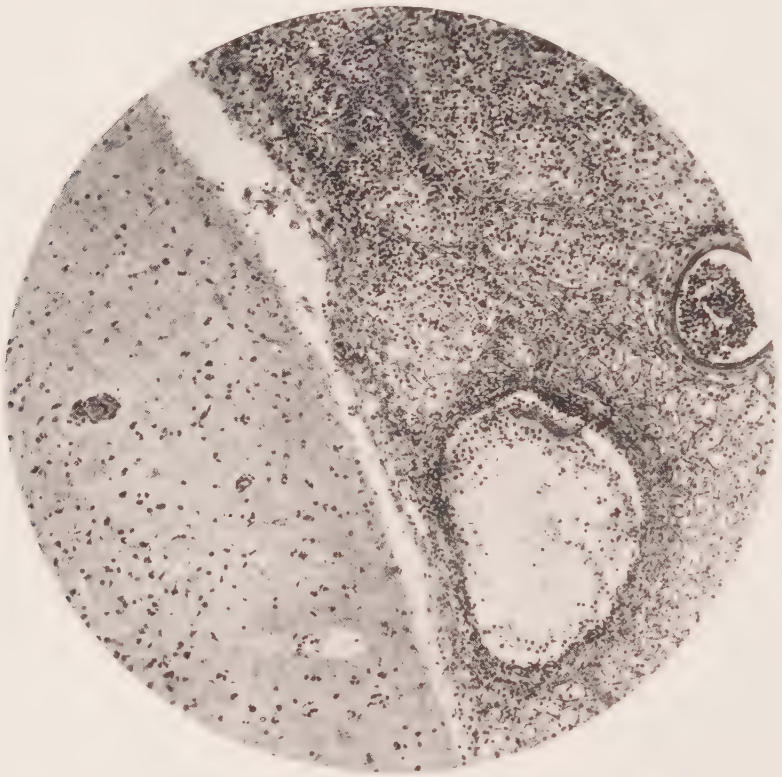


Fig. 915.—Acute suppurative meningitis following suppurative otitis media. × 100. L. H. #232.

the ventricles. It may also occur, as the author saw, for instance, in a 13 year old boy with metastatic staphylococcus meningitis following osteomyelitis of the pelvic bone, as an acute serous, finally suppurative, meningitis with punctate hemorrhages and a serohemorrhagic character to the increased ventricular fluid. The inflammation may spread over the entire cerebral surface in a very short time (see the observation mentioned on p. 1828), usually with the highest degree along the convexity of the cerebrum; or it is at first localized in a situation depending on the point of origin; there are, as a rule, adhesions of the arachnoidea to the dura, usually in circumscribed meningitis. It often spreads in varying degree

to the spinal meninges, particularly in epidemic cerebrospinal meningitis, in which there is marked stiffening of the neck because the exudate is greatest on the base. But a suppurative meningitis coming from the middle ear or the labyrinth may spend its force particularly on the parts in the posterior fossa of the skull, or may even surround the spinal cord throughout its entire length by a thick mantle of pus. Even in twenty-four hours, the spinal meninges to the cauda equina have been known to show suppurative changes following fractures or injury to the skull, as for example, after gun shot wounds (v. Bergmann). In cerebrospinal meningitis the spinal cord is often involved most along its posterior side, particularly in the lumbar region.

Microscopically, the collections of pus cells and fibrin appear in the meninges and the meshes; leucocytes or lymphocytes stream out along the vessels of the pia into the brain substance, where they call forth small encephalitic foci in the cortical zone. Rarely, and usually after traumata, there occur the larger brain abscesses. The etiologically different meningitides, and also the stages of one particular meningitis, vary in the types of cells in the exudates; thus, as demonstrated by lumbar puncture, at first, the leucocytes are the predominant cells; later there appear mononuclear cells, and Speroni calls attention to the increased richness of fibrin in pneumococcus meningitis, and to the large macrophages or phagocytic cells when the neck is stiff (see Busse, Gruber, and particularly Streit). Not only in epidemic cerebro-spinal meningitis, but also in other forms, there are frequent macroscopic encephalitic and spinal suppurative foci with small hemorrhages (Weichselbaum, Kutscher). In fulminating cases, in which the disease leads to death in a few days, the products of inflammation are minor everywhere. In chronic conditions, changes in the vessels of the pia (endarteritis, phlebitis and periphlebitis) step into the foreground (Lowenstein).

Etiology of Suppurative Meningitis.—All suppurative meningitides are called forth by infectious organisms of various kinds. They may be differentiated from the bacteriological point of view as follows: A. Coccus meningitides. These are particularly pneumococci, streptococci (in which case the exudate is usually sero-purulent), staphylococci and meningococci, rarely micrococcus tetragenus (lit. by van Riemsdyk) and gonococci (de Josselin de Jong, lit., Lindenfeld). B. Bacillary meningitides are caused by the typhoid bacillus, colon bacillus, pneumobacillus of Friedlander, diphtheria bacillus (Glaser), influenza bacillus (Pfuhl, lit. Jundel Boyowitsch, lit.), bacillus pyocyaneus (Kossel, Schlagenhaufer), glanders bacillus, the more rare bacillus enteriditis of Gartner (Smith, lit.), various anerobes. C. Meningitis from streptothrix: actinomyces (Wegelin, Seidenberg, lit.). Suppurative meningitis also occurs in malaria

(Durck). It is perhaps more useful, however, to subdivide meningitis into its anatomic forms, thus: 1. meningitis by continuity, 2. metastatic or embolic, 3. primary, the so-called idiopathic meningitis, which occurs in epidemics as well as sporadically; in many cases the condition is secondary to diseases of the middle ear, the nose, or accessory sinuses which are the portals of entry for the bacteria.

(*a*) From infected injuries of the bones of the skull in which suppuration begins in the diploe and is followed by thrombophlebitis of a sinus, or (*b*) from infectious processes in the soft parts of the head such as injuries, erysipelas of the scalp, furunculosis of the neck or face, orbital phlegmons; furthermore (*c*) from suppurative diseases of the pharynx, particularly the nose and the accessory sinuses (for the lymphatic communications see pages 292 and 1829); (*d*) following gun shot wounds of the cerebrum which produce an internal pyocephalus; from here the suppurative inflammation follows the chorioid plexus and the telae chorioidea of the cerebrum and the cerebellum through the foramen Magendii and the lateral apertures into the subarachnoideal space. In these cases, while the meningitis is often confined to the base, it may nevertheless creep to the convexities of the cerebrum and cerebellum (Chiari, the author, and others). Pyogenic organisms may also reach the meninges following blunt trauma (lit. by Ehrenroth, Merkel, Simons), as for example, a fall on the nose with fracture of the ethmoid bone or shattering of the cribriform plate which may also occur in gun shot wounds (v. Hansemann); v. Hansemann saw this occur even through pressure of the atmosphere. In an observation of the author, the base of the frontal lobe was shattered along its surface, and inside of 36 hours a diffuse suppurative meningitis arose with the formation of a large amount of greenish yellow, creamy exudate. The trauma may have occurred some time previous, or, as in the case of Prym, it produced a communication between the accessory nasal sinuses and the meninges. But especially in chronic disease of the accessory sinuses, pyogenic organisms following a blunt trauma may enter the meninges without leaving behind a demonstrable traumatic portal of entry. The author observed a case in a 65 year old carpenter who had been hit above the right eye by a drill; in three days he died of suppurative meningitis; pneumococci were found in the meningeal pus and in the chronically diseased right maxillary and sphenoidal sinuses. A basal leptomeningitis may occur from spread of suppuration of the dura from abscesses of the brain, particularly from an internal pyocephalus following rupture of an abscess into the ventricle (fig. 936).

Particularly important etiological factors in suppurative meningitis as well as in brain abscesses are carious diseases of the bones of the skull, as exemplified by suppuration in the temporal bone (otitis media and

interna), accompanied by caries of its petrous portion (p. 1105). One third of all brain abscesses are secondary to otitic lesions (Korner). As v. Bergmann emphasizes, it is particularly the acute exacerbating chronic suppurative otitis media of fetid character, accompanied by the formation of cholesteatomatous masses and polypoid granulations in the tympanic cavities, that produces meningitis or brain abscesses, since it has a tendency to rupture into the cavity of the skull (p. 1105). See E. Rist concerning the numerous species of bacteria. In addition to the ordinary pyogenic organisms, there are found the pneumococcus, bacillus pneumoniae, and the typhoid and similar organisms. Spread to the meninges or to the cerebral substance follows (*a*) through the tympanic cavity through the tegmen tympani; there occur pachymeningitis and meningitis or the formation of an epidural, epitympanic abscess or an abscess originating probably by way of the perivascular lymph sheaths, but one which is usually separated from the diseased petrous portion of the temporal bone by a thin, only seemingly normal, layer of brain substance. (*b*) In similar manner the inflammation may spread from a suppuration or caries in the cells of the mastoid bone. Most abscesses in the cerebellum arise from this situation. (*c*) Pyogenic organisms are carried by way of the lymph channels along the nerve sheaths of the acoustic and facial nerves and along the vessels in the petroso-squamous fissure. (*d*) A meningitis may also follow infectious sinus thrombosis, particularly of the transverse sinus, which may be accompanied by perisinus abscesses; furthermore, from infectious thrombosis of the smaller sinuses in the petrous portion of the temporal bone, namely the superior and inferior petrosal sinus, and the cavernous sinus. As complications, abscesses also occur under the sterno-cleido-mastoid muscles (Martens).

By the metastatic route, a suppurative meningitis may come from primary conditions elsewhere (occasionally as a single metastasis, see E. Levy), in other cases as the accompanying condition of multiple abscesses in pyemia, ulcerative endocarditis, etc. The meningitides of pneumonia, typhoid fever, scarlet fever, influenza, variola, etc. are either accompanying conditions of the infection, or are mixed infections.

The suppurative meningitis of sucklings is said to be due to the hematogenous invasion of the colon bacillus from a diseased intestinal tract (Sevestre). But the ear or the nose in sucklings may also be points of origin for a colon bacillus meningitis; the bacteria reach the nose and ear in the bath water; small house epidemics have been reported (Scherer). Most of the otitides in sucklings are, however, pneumococcus infections (Netter), with the portal of entry in the nose.

In the so-called "idiopathic" meningitides, which arise sporadically, the most varied pyogenic organisms may be found; at autopsy suppurative

processes can often be found in the nose, accessory sinuses or middle ear, and bacteria similar to those in the meninges are found in these cavities. The diplococcus pneumoniae is a common offender; the streptococcus pyogenes is likewise frequent (Eug. Fraenkel). Formerly the diplococcus pneumoniae (lanceolatus) was believed to be the cause of so-called epidemic cerebrospinal meningitis, and in many epidemics (or perhaps only sporadic cases) this was probably true (Foa and Uffreduzzi). Later it was found, however, that epidemic cerebrospinal meningitis of independent spread, as occurring in garrisons, workshops, etc., but also in general populations, chiefly among the poorer classes (Busse, lit.), was due to an organism resembling the biscuit-shaped gonococcus (Kiefer, Ghon and others), found usually in leucocytes which dominate the exudate (Speroni); this has been called the diplococcus intracellularis meningitidis (Weichselbaum), or the meningococcus intracellularis, or the micrococcus meningitidis cerebrospinalis (Albrecht and Ghon, Weichselbaum). This organism may be cultivated on ordinary agar at body temperature (for particular media, see Felsenreich); for definite diagnosis, special culture media and agglutination are necessary. It is gram negative (see p. 1450); the size of the diplococcus varies; occasionally it is found in the tetragenous forms. In the meningeal pus, the cocci must be identified in isolated leucocytes. The etiological relationship of the meningococcus has now been proved beyond question (lit. in appendix); it may produce a meningitis as well as a septic general infection, one or the other independently, or both together (Graetz and Deussing, lit.). The epidemic spread occurs chiefly in the colder times of the year, and since the meningococci are quite susceptible to cold and dryness, infection occurs probably only through direct contact, and though adults are not entirely immune, it is much more frequent in childhood. The general mortality is high; according to Kirchner, 66%; in children it reaches 90%. Even quite early, the organism can be recognized in the fluid obtained by lumbar puncture (Osler, Councilman and others). It has also been frequently demonstrated, sometimes in enormous numbers, not only in the secretions of the nasal cavity of those having the disease, but also,—a very important finding, in the nasal cavity of the healthy (Jager, O. Heubner, C. Frankel), who are thus "carriers," and may initiate and spread an epidemic. According to Bruns and Hahn, the number of healthy carriers may be 10 to 20 times the number of those having the disease. Since the organism often lives for many months in the nasal cavities of healthy individuals, it can probably be assumed that they are always present in many localities, and produce sporadic cases at certain times of the year, or even call forth an actual epidemic. The figures vary concerning the frequency of meningococci in the blood, but it cannot be doubted that

a meningeal disease is merely an expression or phase of a sepsis (bacteremia); in fact, septic general infection with meningococci has been observed without meningitis (Graetz and Deussing, lit.). The rarer localizations such as in the heart valves, the joints, seminal vesicles (see there), testes, etc. have been described by C. Sternberg (lit.); (see among others, Gruber, Pick, lit., Eug. Fraenkel concerning petechial hemorrhages and other skin eruptions). Portals of entry: according to Westenhoeffer (see also Agazzi, Flexner), the naso-pharynx, according to Goppert and others the entire respiratory tract. Invasion of the meninges follows the lymphatic route from the nasal cavities (probably frequent), or via the blood stream (see also Busse, and Gruber, literature). [For rare meningococcus meningitis following head injuries, see Richey, de W. G., and Helmbold, T. R., Am. J. Med. Sci., 166: 559: 1923.—*Translator.*]

If recovery takes place from a suppurative meningitis, there are likely to remain scarred residues and possibly hydrocephalus.

Chronic Leptomeningitis.—If at autopsy a diffuse or spotted milky cloudiness and thickening of the tender meninges is found, especially along the larger vessels, it cannot be spoken of as a chronic progressive inflammation in most cases. As a rule, it is merely a connective tissue hyperplasia, which may be combined with calcification, as a residuum of a previous inflammation, *e.g.*, a cerebrospinal meningitis, a basal meningitis following suppurative otitis media; or it is the result of stasis and chronic edema, most markedly developed along the veins emptying into the longitudinal sinus; or it develops from as yet unknown causes (toxemia?) in renal disease, alcoholism, or as a result of old age.

In chronic alcoholism, a leptomeningitis pigmentosa, fatty degeneration of the ganglion cells, and overgrowth of the glia are common.

See Lissauer concerning chronic leptomeningitis with perivascular infiltrates of lymphocytes and plasma cells in chronic experimental alcohol poisoning.

The finding of tender, thread-like adhesions between the dura and arachnoidea are quite certain evidences of a previous meningitis, as repeatedly seen by the author following healed suppurative otitis media. An insidious productive inflammation causes numerous, fine, cob-web-like adhesions between the dura and the leptomeninx, particularly in the anterior fossa of the skull; they are found as the brain is taken out; they may produce increased intracranial pressure.

Actual chronic inflammations, consisting of cellular infiltrations and fibrous hyperplasia, occur in the neighborhood of chronically diseased bones, over tumors, abscesses, old degenerative masses, and are usually localized to one place; the pia is often adherent to the surface of the brain.

Furthermore, they occasionally occur in the acute infectious diseases as thick albuminous exudates in the masses of the thickened basal meninges, quite similar to the exudate in periostitis albuminosa, or as cloudy flakes and spots of cell masses in the thickened milky meninges; calcium infiltration is often coincident. Hydrocephalus is frequent in such cases, because the subarachnoideal sinus takes part and is obliterated, or the communications with the ventricles are obliterated. In the progressive paralysis of the insane, there is found almost constantly a chronic leptomeningitis, or better termed, a chronic meningoencephalitis.

See page 1825 concerning chronic serous meningitis.

Infectious Granulation Tumors of the Leptomeninx

Tuberculous Meningitis.—This condition, seen most frequently and in most typical form in children, but also in adults, is, almost without exception, secondary to hematogenous spread; its form is that of tubercles, particularly in the soft meninges, usually accompanied by an inflammatory exudation of thick serous, sero-fibrinous, sero-purulent, fibrino-purulent, or, rarely, pure suppurative fluid. The process always spreads from the pia to the cerebrum (meningo-encephalitis tuberculosa).

In 192 cases (Basle), 67 occurred in the first year of life, 36 in the second, 41 in the third, 22 in the fourth, 14 in the fifth, 9 in the sixth, 2 in the seventh, 1 in the eighth decade; (compare the statistics of Ilzhofer, Steinmeier, lit.).

Disseminated tubercles can also be found in the cerebral substance; thus in the white matter of the cerebral hemispheres, more rarely in other situations such as the cortex, etc.

Sometimes, especially in children, the formation of nodules is predominant; in other cases, particularly in adults, the exudative process is in the foreground.

Origin.—Tubercle bacilli, the bovine type is also quite important (see p. 490), extend by continuity from the neighborhood (bones of the skull, particularly petrous portion of the temporal bone, or the dura), or, more frequently, through the blood stream from another, older tuberculous organ, such as the bones, or the lungs in adults; in children, from either the lungs (H. Koch) or the bronchial, or, more rarely, the mesenteric nodes. The author saw a case (42 year old man), in which, with the exception of a tuberculous meningitis and caseation of the left adrenal, there was no tuberculosis in the body. The condition is relatively frequent in urogenital tuberculosis (see Litten, and particularly Simmonds); according to the author's experience, in 100 cases of urogenital tuberculosis (59 males, 41 females), 33 had tuberculous meningitis (23 males, 10 females), among which there was the relatively high number of 19 (15 males, 4

females) in individuals in the third decade of life, which was probably because in this decade there is particularly vigorous sexual life (see I.-Diss. Rautberd; also the new work of Simmonds which presents similar percentages). In the metastatic forms there may be differentiated: (*a*) disseminated miliary tuberculosis of the meninges which usually leads to death in a few weeks. According to the course, there are differentiated acute and chronic disseminated meningeal tuberculosis. (*b*) Chronic localized tuberculous meningoencephalitis. In the former, the larger vessels are surrounded by innumerable bacilli; in the latter, a few bacilli reach the meninges and call forth a tuberculous meningitis which slowly spreads, and may be confined to a small part of the brain for a long period of time.

Disseminated meningeal tuberculosis is combined quite often with acute miliary tuberculosis in other organs (in ⅓ of the cases; 44.6% Steinmeier; Hartwich 75%). This is particularly frequent in children; but the symptoms in the lungs (see page 429 and following) may dominate the picture. There are, however, cases in which miliary tubercles do not occur in other organs. Occasionally, the disease appears suddenly in otherwise healthy individuals; in other cases it follows a manifest chronic tuberculosis of the lungs, joints, bones, urogenital apparatus, etc. A traumatic tuberculous meningitis is also said to occur (Schultze, lit.).

Disseminated Miliary Tuberculosis.—After removal of the tense, bloodless dura at autopsy, the convolutions of the brain are flattened (increased fluid in the ventricles compresses the hemispheres against the skull), the surface is cloudy, dry (the subarachnoid fluid is pressed out) or is covered with a small amount of cloudy fluid. At the base of the brain, contrariwise, there is found considerable cloudy, gray, thick or milky, or, exceptionally, fibrino-purulent exudate in the markedly injected, cloudy pia-arachnoid, which may show numerous petechial hemorrhages, chiefly in the region of the chiasm and the Sylvian fossa, as well as around the pons, the cerebellum and the medulla oblongata, so that the surface markings at the base of the brain are entirely hidden. The infiltrated meninges feel softer, or tougher, or finely granular. On closer observation in oblique light, after the Sylvian fissure has been exposed, which may be difficult because of adhesions, and after the leptomeninx has been pulled off the brain, taking parts of the cortex with it, there may be recognized the smallest (submiliary), gray white, glassy tubercles, in the exudate as well as in the reddened, but not markedly changed, soft meninges. The tubercles lie in rows along the vessels, more rarely, along the nerves, and produce elongated rounded swellings along them; in part they are scattered promiscuously in the leptomeninx. Nodules are often seen on the dura, isolated, or in groups, among other situations, along the

middle meningeal artery and its branches, and over the clivus. The ventricles are enormously dilated; fluid exudate from the plexus is cloudy or puriform, seldom hemorrhagic; occasionally it contains floating parts of the fornix, the septum pellucidum or the lateral walls. The parts may also be peppered with small punctate hemorrhages. The conditions presented are not merely a passive softening or maceration, but are chiefly the result of edematous swelling, and the softening of an encephalitis (microscopic round cells and fatty degeneration).

Not rarely, the inflammatory exudate disappears macroscopically. In more than half of the cases, however, it can be seen grossly. Microscopically it can practically always be found.

Tuberculous nodules are frequently found in the ependyma, partly on the surface, and partly in the depths (Ophuls, Walbaum). It is necessary to avoid mistaking the ependyma granulations which also occur in this situation. The tubercles are usually larger and unequal in size, occasionally cloudy yellow.

The chorioid plexus is richly sprinkled with tubercles and exudate, and the point of exit of the vena magna Galeni is surrounded by thick granular masses. Because of the edema, the cerebral substance itself appears moist, swollen, soft; very frequently there are hemorrhages (petechial, diapedesis, lit. Biber) and small foci of softening. In cases running a rapid course, there are macroscopic tubercles, but no exudate. As a rule the tuberculous process spreads to the spinal meninges.

The basal parts of the brain on both sides, or only on one side, are preferred, particularly in the typical cases in children; but tubercles may appear, even though sparsely, along the convexities. They are quite frequent and easily seen along the medial surface of the hemispheres; the medial fissure may be adherent. If the term "basilar meningitis" is used, it must be understood that the disseminated tubercles involve chiefly the basal parts, but not exclusively so. Other simple and suppurative meningitides are occasionally localized to the base; as, for example, one following caries of the petrous portion of the temporal bone, or following pyocephalus (p. 1827). Usually, however, in diffuse suppurative meningitis, the convexities are more markedly involved, and in this sense the term "convexity meningitis" may possibly be used. It must be emphasized, however, that the tubercle bacillus, in addition to producing tubercles, may also lead to suppuration, so that tuberculosis of the meninges (particularly in children), and tuberculous suppurative meningitis (more frequent in adults) may be differentiated. In some of the cases, however, there is a mixed infection, *e.g.*, with pneumococci.

The miliary tubercles composed of rounded lymphoid cells, few leucocytes, and epithelioid cells, develop almost exclusively in the adventitia

and the near neighborhood of the blood vessels (small arteries and veins, see Askanazy); occasionally they are so thickly scattered that they obscure the coats. The remaining coats, chiefly of the middle size and small arteries, are always, of course, severely coinvolved; the media shows chiefly necroses; the intima, subendothelial granulation tissue overgrowth, which may lead to obliteration of the vessel; and there is a diffuse small cell infiltration in the veins. Thrombosis may follow rupture of tubercles through the walls (Biber, lit.). The tubercles, in which the bacilli may be demonstrated, usually progress rapidly to caseation. In cases running chronic courses there are large cell epithelioid conglomerate tubercles with giant cells, or a cellular fibrin tubercle. In most cases the appearance of tubercles is accompanied by inflammatory exudation into the meninges

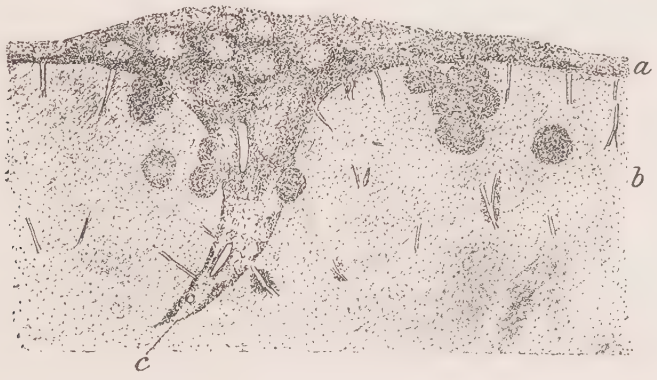


FIG. 916.—Tuberculous meningitis, duration weeks; chronic disseminating tuberculous meningo-encephalitis; a, pia-arachnoid tuberculous and infiltrated by cells and fibrous tissue; b, cerebral cortex with some tubercles; in places perivascular cell infiltration as beginning of tubercles; c, sulcus with cross sections of vessels. (Very low power.)

(from the subarachnoideal space), the exudate composed of a coagulable fibrin-containing fluid and cells. According to Speroni, the mononuclear cells are predominant among the latter, whereas in the acute forms, the lymphocytic cells predominate, and may be demonstrated in lumbar puncture fluid (O. Rehm). They may be so numerous and so thickly scattered that the tubercles can scarcely be differentiated from the exudate. According to Gehry, the pia is infiltrated by lymphocytes, plasma cells and epithelioid cells; see Ranke, Sawada, Wolff, who have described plasma cells. The presence of many large cells in the exudate, particularly in the later stages, is explained by Birch-Hirschfeld who believes there is growth and desquamation of the endothelial cells from the lymph spaces, and, further, that this is an effect of the absorption of tuberculous virus from the previous vascular tuberculous foci. Orth, Speroni emphasize, however, that these may be mistaken for macrophages of the large, usually mononuclear, cell type, which come from lymphocytes and which may also

appear in other forms in meningitis (p. 1827). (Concerning macrocytes in general, see volume 1, page 9.) According to v. Fieandt, the large cells in the exudate come from lymphocytes, and from fixed tissue cells. Wiethold (lit.) believes the large cells which he calls histiocytes, are of connective tissue and endothelial nature. Spielmeyer also believes the macrocytes are histiocytes, which must be differentiated from true lymphocytic cells. (Concerning histiocytes, see volume 1, pages 9, 196, 220.) According to Mandlebaum, numerous mononuclears which contain tubercle bacilli can be demonstrated in the lumbar puncture fluid obtained at post mortem, and he believes they are still living cells coming from the meninges. Following the sheaths of the vessels in the pia, the

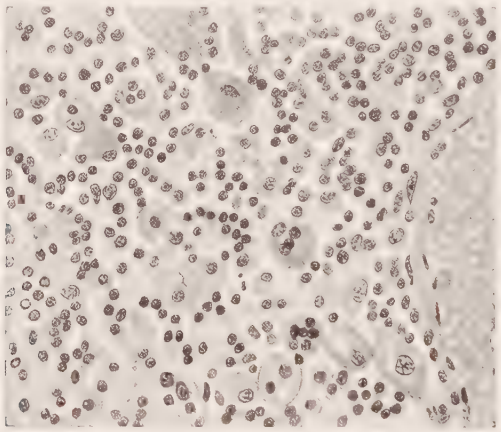


FIG. 917.—Large cells, *i.e.*, mixed macrophages and lymphocytes in cerebral sub-arachnoid space in tuberculous meningitis; boy 12 years old (high power).

inflammation spreads and becomes an encephalitis or a neuritis of the brain substance itself, or of the cranial nerves. Following endovasculitis, edema is very frequent in the cerebral substance; the nerves swell and later undergo destruction in the form of focal softenings which may be ischemic, or, more rarely, hemorrhagic (Nonne, Vanzetti). Bacilli are often found in the lymph sheaths. Tubercles are frequent in the perivascular sheaths, and may be found in the lymph spaces. The consequent lymph stasis on the one hand, the vascular position of the tubercle on the other hand, the exudate saturating the meninges and the edema of the cerebral substance itself, readily explain the severe circulatory disturbances suffered by the patient. Hemorrhages in the medulla and cortex are frequent, and according to Biber (lit.), Kirschbaum and others, they are ring bleedings, *i.e.*, annular hemorrhages around an axial capillary or precapillary vessel; the free space around the vessel is caused by the removal of the red blood corpuscles through the stream of the transudate; according to Dietrich, the hemorrhage does not occur through the primarily injured fibrinous and

necrotic parts of the vessel, but through diapedesis from the vessel in a prestage of the above described changes. (For other explanations, see Oeller, and more details by Spielmeyer.)

The disease is practically always fatal. But cases have been described in which healing took place with fibrous changes around the tubercles and scar-like thickenings in the meninges (lit., by Brooks and Gibson, Krause, v. Bokay, Harbitz, lit.). A rare condition is manifested by scar-like fibroses or thickenings occurring in chronic tuberculous inflammation of the meninges (Busse, Rossle).

Chronic Localized Tuberculous Meningoencephalitis.—When bacteria reach isolated branches, or even one branch of a meningeal vessel, there may develop a local tuberculous meningoencephalitis running a chronic course. There are formed large cell tubercles, which contain many giant cells and which spread into the soft meninges, but also follow the pial vessels into the brain substance and produce large caseous conglomerate tubercles (the so-called solitary tubercle). Such cases of local meningoencephalitis tuberculosa chronica, in contrast to the acute disseminated forms, produce clinical signs of focal disturbance (see under brain tuberculosis, p. 1914). Occasionally, there is observed an encephalitis, or better, encephalomalacia, softening of the brain, as a result of vessel thrombosis, *e.g.*, in a branch of the Sylvian artery, with consequent hemiplegia or more localized focal symptoms (Weintraud and Biber, lit.); but this may also occur as stated on p. 1836, tuberculous meningitis.

See under syphilis of the brain for syphilitic, chiefly gummatous, meningitis, p. 1916.

Actinomycotic meningitis is rare. The exudate is fibrino-purulent, and fills the subarachnoid meshes; in the deeper layers of the pia and in the walls of the veins are many lymphocytes and plasma cells. It arises either through direct continuity from the neighborhood, or via the hematogenous route, particularly from actinomycosis of the jaw and of the remaining parts of the face, middle ear or of the lungs. Rarely, it is primary. There occur pure and mixed infections. Localization: chiefly the base of the brain and the spinal meninges. Superficially, the condition may resemble epidemic cerebrospinal meningitis. Spinal puncture obtains a fluid rich in leucocytes and perhaps actinomyces. In rare instances, actinomycotic meningitis is latent clinically. (For more details and lit., see Wegelin, Geymuller, Stahr.)

Tumors and Parasites of the Soft Meninges, the Telae Chorioidea, and the Ventricles (Lit. in Appendix)

Primary tumors (*a*) in the meninges are not frequent; they are usually sarcomas or endotheliomas and may take the form of (1) one single large

tumor, or (2) multiple small or large, or more rarely (3) a diffuse, flat, spreading tumor in the cerebral and spinal meninges; they arise in part from the endothelial cells which cover the meshes of the soft meninges and in part from the adventitia of the vessels as peritheliomas (Hart, endothelioma perivasculare, and Lewy; Haeger). The tumor masses are composed chiefly of islands of flat cells, concentrically layered, similar to a squamous epithelioma, and embedded in connective tissue, or the cells are more cylindrical in shape and there is produced an endothelioma lymphangiomatosum-like tumor as described on p. 179, and resembling a

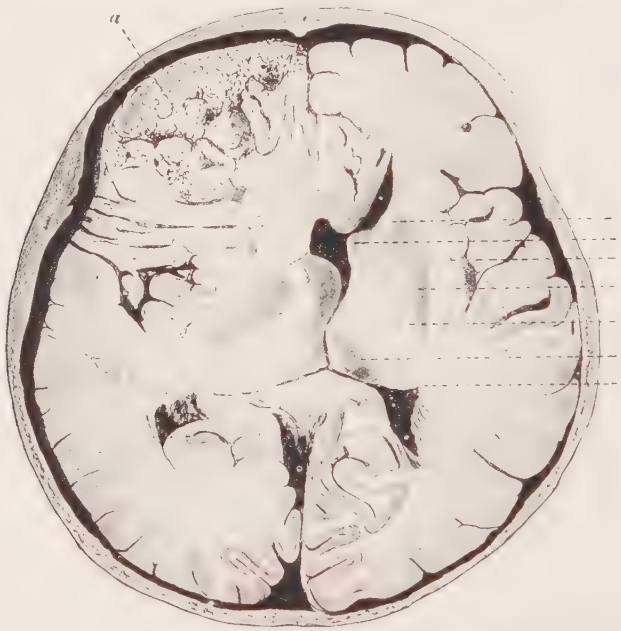


FIG. 918.—Tumor of the pia-arachnoidea, vascular round cell sarcoma (much hyalin degeneration of vessel walls) in area of r. frontal lobe (a); marked distortion of architecture by tumor pressure; woman 59 years old. Brain and skull cap cut at usual plane (about a finger's breadth above orbital margins and auditory orifices); view of upper cut surface; Ci, internal capsule.

cylindrical cell carcinoma. Other sarcomas are hemangiosarcomas, in which hyalin change may take place in the vessels producing cylindromatous structures (illustrated under spinal cord). Ordinary sarcomas also occur, *e.g.*, the round cell type, in which there may also be hyalin change in the vessel walls with hemorrhages, myxosarcomas, polymorphocellular sarcomas, etc. Myxomata arising from the arachnoidea are very rare, as are diffuse sarcomatoses coincident in the spinal meninges (Nonne); this has been observed in melanosarcoma (Albrecht, Bosch, lit.). Primary multiple sarcomas of the leptomeninx, of the brain and spinal cord appear in childhood as well as later (lit. Rach, Kaiser); metastases are, however, quite unusual (Olivercrona).

Cholesteatomas—epithelial new growths containing cholesterin crystals—are quite uncommon. They usually occur at the base of the brain, more rarely in the ventricles, or even in the spinal cord (Chiari); they present waxy white, soft, rounded or nodular masses with the sheen of mother-of-pearl; they are often joined in conglomerate masses, are quite brittle to the touch, or splinter on slightest handling. Their size varies between that of peas and apples, but cases have been observed in which the tumors reached the size of a fist and compromised neighboring structures. They are almost always connected somehow to the vascular pia, either lying upon it, or connected with the chorioid plexus; they may press deeply into the substance of the brain, since the subarachnoid space is no longer large enough to accommodate them. Their sites of election are the neighborhood of the pons, medulla oblongata and cerebellum, and the space between the olfactory convolutions and the corpora candicantia (Bostroem), occasionally in the ventricles. Microscopically, they are composed of a thin fibrous wall supplied by the pia, the inner surface of which is covered by a layer of flat, polygonal cells whose epidermoid origin is recognized by the keratohyalin granules, and by cell masses desquamated from the wall, and a material composed of plaques and plates of cholesterin appearing during the destruction of flat epithelium. Ziegler found hair in such tumors, Bonorden found sebaceous glands and hair, and in such cases there may be skin lying upon the pia. Formerly it was assumed they were of endothelial origin from the lining cells of the subarachnoid connective tissue spaces (Virchow), a viewpoint which Borst adopted, but in addition believed that the perithelium of the pial vessels might also be responsible for the tumor, (see also Durck); but others, particularly Ziegler, believe it is an epithelial structure of epidermoid origin, an opinion confirmed by the investigations of Bostroem. With Gram-Weigert's staining method, keratohyalin granules may be demonstrated in the cells. For the great majority of cases, it is probably correct to assume there was an early snaring-off of epidermis, in which event there arise dermoids, or dermoid cysts as large as goose eggs with skin, glands, hair and erectores pilorum and a lamellated content surrounded by connective tissue. The snaring-off furthermore need not take place from the anlage of the hypophysis, but germinal epithelium from the region of the head may occasionally be snared-off from the cerebral vesicle at the base of the brain. There may, however, also arise cholesteatoma in the subcutis, the pericranium, the bones of the skull (lit. Orlandi) and the dura. Most authors believe in Bostroem's conception; compare Thomas and Erdheim (lit.), Scholz, Binder-Schwalbe, Frick and others. Since the tumors do not occur in the midline, but in a lateral position, Teutschlaender offers the suggestion that these dermoids arise from germinal cells misplaced during

the formation of the inner ear. Ernst saw a carcinoma arising from such a structure. See F. Stromeyer (lit.) concerning combinations of cholesteatomas and sarcomas. Occasionally, the pulp of a dermoid cyst is emptied into the subarachnoid space (Erdheim).

Cavernous hemangiomas are rare; they produce diffuse growths of the character of telangiectasias, or an angioma arteriale racemosum (for example, the case of Kalischer); see also under brain, p. 1933. Cavernous

FIG. 919.—Cholesteatoma of base of brain of man 26 years old; part of tumor removed by operation; located in right cerebellar cornua of pons. To left are pons, trifacial, acusticofacialis, glossopharyngeus, abducens; asymmetrical vertebral arteries; adhesions over basilar artery and between the oculo-motor nerves, the int. carotid artery and the optic chiasm; nat. size.

lymphangiomas (p. 177) are still rarer. Lipomas are uncommon, but occur in the region of the base of the sphenoid bone and the corpus callosum; their growth and spread is probably dependent upon the development of the convolutions. Ernst described a lipoma containing a few spicules of bone, lying along the convolutions, as mentioned by Virchow. The collection at Basle contains a similar preparation, with partially agenetic convolutions (von Sury). Huebschmann saw a "convolution lipoma" with partial defect of the convolutions. Bostroem emphasizes the close relationship of lipomas to the pia, and believes they have a point of origin similar to cholesteatomas, namely, an ectodermal misplacement

of germinal cells, in which, however, of the entire dermal anlage, only the connective tissue parts grow in the form of fatty tissue. (Lit. by Zuckermann, lipoma at the tuber cinereum; Hecht, lipoma of the left Sylvian fossa; Nippe, lit., Gautier.) Villous fibromas, osteomas are quite rare. (But the formation of small nodules of bone in the meninges is not rare, particularly over the frontal lobes, and in the leptomeninx of the spinal cord.)

From the chorioid plexus there may arise benign and malignant epithelial tumors. The former, (frequently observed, see Bruchanow, Vonwiller, lit., Borchers, lit., Natonek, lit.), may take the form of a papillary growth (the so-called plexus epithelial papilloma) with a covering of cylindrical epithelium in which the stroma may become mucoid and the stalk of the papilla cystic. The epithelium is simple cylindrical, glandular, or ciliated as in the embryonal plexus, in contrast to the ependyma epithelium. Complicated gland-like arrangements resemble "adenomas," and Bielschowsky calls them so, since he vindicates the character of the plexus epithelium as a glandular epithelium, and especially since it is also assumed that the plexus epithelium and even the different ependyma epithelium (?) secrete the cerebrospinal fluid; Milan, indeed, terms the plexus a gland, interposed between the blood and the cerebrospinal fluid. Carcinomas (lit. Korner), likewise of papillary structure, may invade the neighborhood and produce secondary nodules. Benign and malignant tumors may also arise from the connective tissue and the vessels; for example, fibromas, which through the deposit of calcium concrements become psammomas; less frequently, lipomas; hemangiomas, among them those with hyalin changes (cylindromatous); enchondromas originating from parts of the primordial cranium; osteomas as well as gliomas, carcinoma-like neuroepitheliogliomas (Korner), a perivascular sarcoma (Watzold), (Hunziker, lit.).

Frequently there is an increase in the brain sand in the plexus as well as multiple cysts; and plexus cysts with watery or colloid contents are manifested as edema in the soft connective tissue stalk of the villus (Askanazy). The plexus may appear like a bunch of grapes. Plexus cysts in the third ventricle are rare and produce severe symptoms; the cysts may be as large as a cherry and obstruct the ventricle. The author saw a case in a 43 year old man, in whom, because of varying degrees of increased intracranial pressure symptoms, a brain tumor was diagnosed; the author saw a cyst as large as a cherry stone inserting in the tela chorioidea behind the anterior commissure in the third ventricle; the hypophysis was flattened, the sella turcica was softened. There was marked hydrops of the lateral ventricles. The hands and feet were very large, the thumbs particularly broad, (compare cases of Sjovall, Beutler, Busch, Bittorf). In a 17 year

old girl with complete blindness of the left eye and beginning blindness of the right, the author saw a cyst of the plexus as large as a billiard ball, situated in the inferior cornu of the lateral ventricle on the left side; the intracranial space was markedly diminished; extensive calcium metastasis in the dura.

The so-called cholesteatomas of the ventricle plexus in humans, which may reach the size of beans and are a yellow white, rounded, tumor-like inclusion similar to the larger tumors of horses (for more details see Joest; see also Saul), are not true cholesteatomas, but infectious granulations with embedding of doubly refracting substances in the cells (the so-called pseudoxanthoma cells) and of cholesterin needles, about which are grouped foreign body giant cells (granuloma cholesterinicum, Schmey).

FIG. 920.—Glioma of IV ventricle (columno-cellular gliomatous neuroepithelioma, see text) derived from the posterior medullary velum in man 22 years old. Drawn by author from fresh specimen.

On the inner surfaces of the ventricle. The granules to be described later under ependymitis occasionally lead to the formation of small papillary tumors. Not uncommonly, there are found ependymal gliomas as small, broad-based, pedunculated, usually solitary, tumors as large as peas or larger, of tough consistency and occasionally present in all the ventricles (Henneberg). They are composed mostly of many small cells, and are without apparent connection with the ventricular epithelium, even though there do occur isolated spaces lined by epithelium (lit. in appendix). There occur, however, other forms of gliomas in the ventricles which reach the size of walnuts or hen's eggs (see ventricular tumors under tuberous sclerosis, and p. 1908 and fig. 949). The gliomas of the fourth ventricle take on a particular type, and often fill this cavity like a fungus. The author described such a case (fig. 920) occurring in a 22 year old man, (published in detail by Muthmann and Sauerbeck, lit.). The tumor arose from the velum medullare posterius and was composed of neuroepithelium (ependyma epithelium and ependyma cells) in columnar arrangement,

encircling blood vessels (neuroepithelioma gliomatosum columnocellulare). Roman described a histiogenetically similar, but malignant tumor arising from the ependyma of the third ventricle in a 21 year old girl (situation third ventricle and toward the pineal body). In the ependymal glioma of the fourth ventricle described by Link, the basic tissue was a papillary glioma composed of fibers of glia grown through and covered by ependymal cells, some of which were in glandular formation. Other richly cellular, poorly fibrous gliomas, the so-called glio-sarcomas, of all the ventricles (Hildebrandt), and ordinary sarcomas of round cell character which may, however, easily be confused for richly cellular gliomas, are very rare (lit. by Hunziker). Papillary epitheliomas and carcinomas arising from the ependyma epithelium of the ventricle are also infrequent, (they may be of papillary or glandular structure). The author examined an adenocarcinoma coming from the lateral ventricle, which microscopically was quite similar to carcinomatous intestinal polypi; the epithelium of the gland imitations was exquisitely cylindrical; many cells contained colloid droplets, which also lay in the lumena; in other situations they were polymorphous and stratified. The connective tissue was present here and there in the form of plump papillae jutting into the lumena of the glands. Just like the stroma of the papilla, it was in part finely meshed and myxomatous, and in part granular and necrotic. There were also many unequally thick, convoluted, epithelial glandular spaces and cylinders which probably were lying free in the surrounding soft tissue masses. v. Wunschheim and Saxer (lit.) described a similar case, in which the ventricular ependyma, as well as the plexus epithelium, were believed to be points of origin (likewise in the case of Bondet and Clunet, lit., Hart, lit.). In addition to cylindrical cells, there were squamous epithelial cells in a case described by Ziegler. Selke found squamous epithelium in a papillary fibroepithelioma of the third ventricle, and is inclined to believe that the tumor came from epithelium of the mouth cavity, an opinion which relates this tumor to those arising from the hypophyseal ducts. See Vonwiller, also Korner, concerning the epithelium of the ventricles, and a critique of the tumors arising in them. See Saxer (lit.), Sjovall, concerning teratomas of the ventricle.

Melanoma is a name given by Virchow to tumor-like pigmented growths in the pia-arachnoidea, found in places where the normal brown pigment nodules rest in the larger branched or stellate connective tissue cells (or chromatophores) of the internal layers of the pia; in most adults these are found along the ventral surface of the medulla oblongata (for more details, see Broniatowsky). The pia may be entirely brown, and occasionally smoky gray or black. For good microscopic pictures it is necessary to use fresh, teased preparations. This melanosis must not be

mistaken for the residues of hemorrhage. Pigmentation also occurs in other situations along the base of the brain, as, in the region of the olfactory bulb, and in the fissure of Sylvius; furthermore, pigmented cells may be found in the adventitial sheaths of the vessels of the brain and spinal cord. The observations of Virchow were confirmed, among others, by Stoerk, and it is now generally accepted that melanotic sarcomas composed of spindle, or large round cells, and which often spread diffusely over the cerebral and spinal meninges (melanosarcomatosis), may arise from these cells (lit. by Esser, Thorel, Schopper; see also Pick and Berblinger). Secondary disseminated sarcomas are very rare. In a case described by the author, there was rupture of a telangiectatic myxosarcoma from the medullary layer into the posterior cornu; in both lateral ventricles, the third, and even the fourth ventricles, were small isolated metastatic tumors with the character of the main tumor, which probably arose through implantation of particles of the latter.

Secondary Tumors.—There occur sarcomas, gliosarcomas (Grund), and carcinomas (lit. by Krasting, Heinemann). The usual mode of spread is through the blood stream. The primary carcinoma is frequently in the stomach (Schwarz and Bertels, lit.). Occasionally the metastases spread diffusely along the meninges and following the perivascular lymph channels diffuse, not only into the brain substance itself (Merzbacher), but even into the spinal cord; they may produce symptoms of meningitis. Occasionally, free cells are found in the subarachnoid space (Westenhoeffer, Scholz, Hoffmann and others); Lohe found carcinoma cells under the ependyma; Pachantoni saw focal meningeal hemorrhages. The term "carcinosis" of the meninges is used when inflammatory manifestations are lacking; when these are present as a fibrino-serous or a suppurative exudation, the condition is spoken of as "meningitis carcinomatosa." Eichhorst emphasizes that the carcinomatous infiltration may escape the naked eye, and be hidden under the picture of mild cloudiness, or a leptomeningitis chronica, or an edema, or a suppuration. The author confirms this statement and extends it to include the spinal meninges. Possibly, carcinoma cells, as for instance, from a carcinoma of the stomach, may reach the cranial cavity through the lymph sheaths of the nerves (Knierim, lit.), similar to the conditions seen by the author in suppurative inflammation in a child with empyema of the pleura, in which the suppuration was spread through the intercostal nerves to the spinal membranes. In the centrifugalized spinal fluid, there have been found large, pale, rounded cells with small nuclei, as well as small cell complexes which have made possible the clinical diagnosis of carcinomatous meningitis (Stadelmann, lit.). Carcinoma may be mistaken for primary diffuse endothelioma, or occasionally for chronic leptomeningitis (Saxer and others).

Among **animal parasites, cysticerci** (p. 848) are frequently found in the subarachnoid space and also between the pia and the cortex with preference for the convexities; they often appear in great numbers, in part as transparent vesicles, in the water clear contents of which the head of the animal may be seen hanging, and partly as encapsulated and calcified nodules. According to Marchand, they may reach the smaller arteries of the pia and be distributed through the smaller branches leading into the cortex. It is necessary to remove the soft meninges to detect the cysticercus. The vesicles may spread to the cortex of the brain, while the surroundings are simply compressed, or softened, or sclerotic. Giant cells collect around older nodules (Askanazy). The vesicles remain attached if the soft meninges are pulled off, and there is left behind a spherical or hemispherical space in the cortex. At the same time there

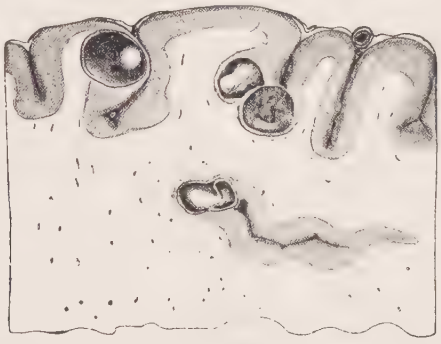


FIG. 921.—Piece of brain tissue showing numerous cysticerci within meninges and substance; the cysts in which heads are visible, force themselves into the cortex; nat. size.

may be isolated cysticerci deep in the cerebral substance, usually in the gray matter and in the ventricles (p. 1852). The soft meninges may be somewhat thickened. Very rarely there appears, as described below under cysticercus racemosus in which this picture is oftener observed, a basal fibroplastic membrane with markedly dilated blood vessels, in which are embedded cysticerci, mostly destroyed; the convexities are free (Kufs). Cysticercus, secondarily adherent to the dura, may remain attached to that membrane when it is pulled off and may be mistaken for a gumma or tubercle. Occasionally cysticerci produce brain symptoms. (See p. 1938 and literature in appendix.)

The cysticercus racemosus (Zenker) is quite rare; it appears in the subarachnoid spaces of the base of the brain and more rarely in the ventricles (Stern, lit.); it is composed of a mulberry or grape-like lobulated and branched vesicle, which, like a veil, covers the larger vessels in the chiasm, and is particularly prone to occupy the Sylvian fissure.

The entire structure represents one cyst with closed daughter cysts, and may be called a cysticercus multilocularis (Marchand, lit.). The scolex is often difficult, or quite impossible, to find.

The situation occupied by the parasite may be the site of a chronic fibroplastic meningitis, which leads to tough thickenings easily mistaken for syphilitic meningitis; the arteries enclosed within, as Askanazy emphasizes, may show peri- and endarteritis—similar to that of syphilitic arteritis,—and even necroses of the walls, and thrombosis (Gross, Kocher). Areas of softening, even of quite considerable size (Gross, lit.), and hydrocephalus may develop. Non-characteristic clinical symptoms of increased intracranial pressure with papillo-edema (less frequent) (Jacoby, Kruger) are much more frequent than in ordinary cysticerci (Risenblath, Wollenberg, lit., Henzler, lit.).

See under parasites of the cerebrum for echinococci.

Blastomycosis is rare and interesting; the meninges, or even the ventricles, have a clouded appearance; cystic or tumor-like structures have a muco-colloid appearance; they are composed of blastomyces, some contained in macrophages (Benda). The portal of entry is possibly (?) the mouth or the pharynx (Busse, v. Hansemann, Turk).

DISEASES OF THE VENTRICLES

Ependyma Epithelium and Plexus Epithelium. The Cerebrospinal Fluid.—The inner surfaces of the ventricles are covered with ependyma epithelium. The chorioid plexus, covered by epithelium, dips into the ventricles; the cubical to polyhedral plexus epithelium, which is a true epithelium coming from the combined anlage of the medulla, is said to have secretory functions; it is supposedly the chief source of the cerebrospinal fluid (Schlaepfer, Goldmann and others); on the other hand, it also has absorptive functions as shown experimentally by Askanazy (lit.) and Klestadt by means of carmin, fat, and glycogen. The ependyma epithelium is different from this genetically, morphologically, and physiologically; according to Benda it is a pseudoepithelium, similar to the spongioblasts, which are the forerunners of the glia, and is anchored in its glious space by a basal prolongation of glia; it possesses, even into old age, a ciliated border, while such structures are found in the plexus epithelium only in embryonal times (Vonwiller, lit.). It probably has secretory, (Fuchs, and others), and resorptive functions (Klestadt); but the latter, under pathological conditions, is quite different from that of the plexus epithelium (Askanazy).

According to Fleischmann, the cerebrospinal fluid is a filtrate from the blood, but the specific activity of the plexus cells removes all injurious substances. (Pathology of the plexus, brain tumors, meningitis, encepha-

litis, etc., by Saito.) See E. Schwarz concerning xanthochromia of the fluid and its significance in the diagnosis of hemorrhages of various kinds.

Monakow may be consulted concerning the circulation of the cerebrospinal fluid, which passes from the ventricles into the walls of the hemispheres, and after it nourishes the nervous part, passes through the perivascular spaces to the subarachnoid space, and then through the venous plexuses, the sinus, Pacchionian bodies into the venous system. Jacobsthal recommends puncture of the foramen magnum to obtain cerebrospinal fluid from the cadaver.

Internal Hydrocephalus (fig. 922)

(External hydrocephalus is edema of the pia-arachnoidea, *e.g.*, over atrophic areas of the cerebral surface.)

Internal hydrocephalus is an accumulation of too much fluid in the ventricles; it is usually watery and quite similar to the cerebrospinal fluid,

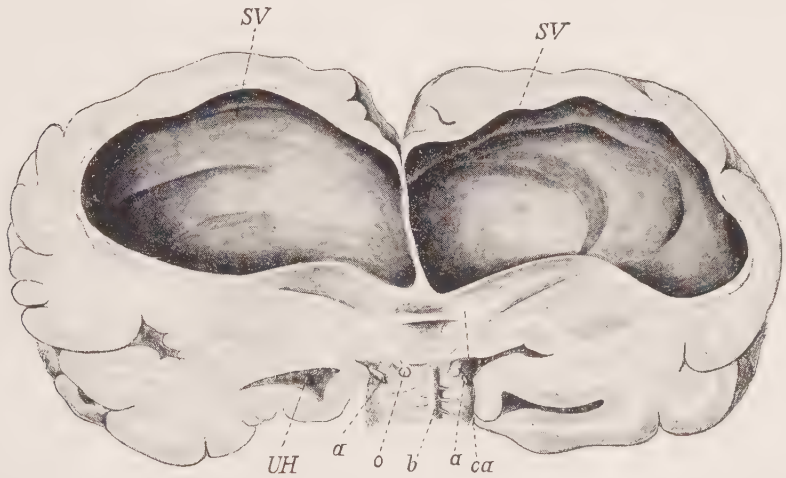


FIG. 922.—Hydrocephalus internus; frontal section through brain, hardened in chromic acid. SV, lateral ventricles; UH, right lower cornua; Ca, anterior commissure; a, mid. cerebral artery; b, basilar artery; o, optic nerve; from an adult male with macrocephaly; ½ nat. size.

but in some cases, it is cloudy like an exudate. According to the composition of the fluid there are differentiated simple and inflammatory hydrocephalus; the exudate in the latter is milky or purulent, and is produced from the richly vascular plexus chorioidea and the ventricular epithelium (lit. by Raubitschek, Yoshimura, see p. 1805), (pyocephalus); it is usually of acute onset; occasionally a simple hydrocephalus takes on an inflammatory character. The fluid accumulates particularly in the lateral ventricles; yellow streaks of pus may cross the veins in the floors of the ventricle. These cavities are thus stretched, their floors are flat-

tened, and the convolutions are pressed flat against the inside of the skull. The septum pellucidum and the commissures are pulled apart. A congenital and an acquired hydrocephalus may be recognized, and among the latter, those which appear in childhood, and those which appear in adults.

Congenital hydrocephalus may be present at birth to such marked degree that it produces obstruction to labor; the head of the child has been known to burst during birth, or while still in the uterus; more often, however, it is of minor degree at the time of birth, but soon increases. The skull is stretched (macrocephalia), and in the first year of life may reach the size of a man's head or larger; the author, for instance, estimates the circumference of an adult head at about 72 cm. The stretched bones of the skull may become as thin as paper, transparent, and show membranous spaces. The fontanelles remain open for years, the sutures gap. When the stretching ceases, there may appear hyperostoses of the skull, with the subsequent appearance of numerous Wormian bones.

See R. Meyer concerning these Wormian bones and defects in the bones of the skull in hydrocephalus.

The face is unnaturally small in relation to the distended, rounded head, (the rachitic skull is angular, box-shaped). The cutaneous veins, particularly in the anterior part of the skull, are wide and convoluted. The ventricles, especially the laterals, contain a clear serous or a cloudy albuminous fluid, the latter when hydrocephalus occurs from inflammatory processes. The amount of serous fluid varies between 50 to 100 cc., and many liters. The pale substance of the hemispheres may be thinned to several millimeters, or less, so that merely the meninges and a very small amount of paper-thin cortical layer surround the saccular spaces which appear like protuberant bladders. At first, the stretching is at the cost of the medullary substance, namely, the corpus callosum, which may have completely disappeared (Anton). The ependyma of the ventricles may be tender or thickened and granular; in inflammatory, rapidly appearing, hydrocephalus, the internal surfaces of the ventricles are quite soft (hydrocephalic softening). Parts of the cerebrum, cerebellum, the pons and the medulla oblongata are pushed toward the vertebral canal or are pressed into this opening (Chiari). Under these conditions lumbar puncture may obtain no fluid (Quincke, Nolke). But the cerebellum may be wedged into the foramen magnum in other conditions which produce increased intracranial pressure such as tumors, tuberculous meningitis, and conglomerate tubercles (see fig. 941); and since Schwalbe found the cerebellum pushed into the foramen magnum in normal brains also, care is necessary in diagnosis. The hydropic dilatation may involve all the ventricles following closure of the foramena of the fourth ventricle, or may be localized to one ventricle, or to a part of one, particularly to the

posterior cornu which becomes cystic. These are often the cases suffering trauma during childbirth (v. Haberer). In unilateral ventricular hydrops, the foramen of Monroe, which connects the lateral ventricles, is obstructed. An isolated hydrops of the fourth ventricle and the ventriculi septi pellucidi also occurs. Occasionally, there are coincident anomalies of development, such as hare lip, cleft palate, club foot, cyclops, encephalocele, spina bifida, dwarfism and others. The duration of life in congenital hydrocephalus is much diminished, and most individuals die shortly after birth or during their first month or year. Some few grow older, but the changes must be less marked; the absolute weight of the cerebral substance may be unchanged. In cases of the latter kind, even the psychic functions may be entirely uninfluenced, but usually children with marked hydrocephalus are idiots or morons. Genetically and etiologically, congenital hydrocephalus is still a puzzle; when the foramena of the fourth ventricle are closed, dilatation due to stasis is quite possible; in the majority of cases, however, all the foramena, even that of Monroe, are markedly dilated (Graf Haller, lit.). There have been postulated anomalies of secretion, increased cerebrospinal fluid or inflammatory exudation from out the plexus and the ventricular epithelium, which have been brought about through toxic or infectious causes (alcoholism or lues of the parents). A familial predisposition has also been postulated. (Lit. by Heubner and Eichmeyer and particularly, Anton; see also Kausch, Motzfeldt.)

Acquired hydrocephalus is differentiated into acute and chronic forms. The acute, which usually is less marked, but because it arises quickly, gives more severe clinical symptoms, is met in tuberculous meningitis so frequently that we use the term acute hydrocephalus and tuberculous meningitis interchangeably; but it also occurs in general suppurative meningitis following rupture of a brain abscess into the ventricles, suppuration following spina bifida, etc. There also occurs an idiopathic, acute, non-tuberculous hydrocephalus. Chronic acquired hydrocephalus appears in later childhood or in adults.

Etiologically, there may be considered: inflammations leading to an increase in the amount of cerebrospinal fluid such as chronic inflammatory changes in the plexus, the walls of the ventricles, and the soft meninges (inflammatory hydrocephalus); serous meningitis is said to follow alcoholism, traumata, acute infectious diseases, etc., (Quincke). Occasionally the character of the fluid, or thickenings in the meninges or plexus point to a previous inflammation; this may have produced an obliteration of some of the subarachnoid spaces and also of the arachnoid villi. In some cases, there is said to have been obstruction in the communicating passageways between the fourth ventricle and the subarachnoid space, but this is doubted by Schmorl; if it did occur there would certainly be disturbance

in the outflow of the cerebro-spinal fluid. (Compare Anton and von Bramann concerning the treatment of hydrocephalus, and of inoperable tumors by puncture of the corpus callosum, which increases the means of communication between the ventricular fluid and the subarachnoid spaces; Hollenbach, lit.) The suboccipital puncture serves the same purpose, *i.e.*, reducing pressure through drainage of the fluid; this consists in a median opening of the membrane of the atlanto-occipitalis and the dura (Anton and Schmieden); for clinical remarks, see Eden. (Literature by Propping, concerning the mechanics of the fluid.) Furthermore, disturbances in the outflow of the cerebral spinal fluid may be caused when the aqueduct of Sylvius is closed by a tumor or a membrane (Wolf, lit.), or by warty and ependymal growths at the entrance of the aqueduct, such as described by Verse, or by a tumor in the pons or the cerebellum which pushes the tentorium upward and thus compromises the aqueduct and also the vena magna Galeni. Thus in a cystic glioma of the superior vermiform process, (6¼ year old boy), the author found 2500 ccm. of watery hydrocephalic fluid; the circumference of the head was 66 cm. Other cases appear from disturbance of the venous outflow (stasis, hydrocephalus), *e.g.*, through pressure of a tumor in the cerebellum on the vena magna Galeni. Primary chronic internal hydrocephalus in adults is believed by Margulis (consult for details) to be due to a chronic peri-ependymal glio-fibrosis, which is caused by intrauterine damage to the anlage and to the development of glia. This glio-fibrosis of the ventricle hinders the outflow of fluid and thus is said to cause dilatation as the fluid accumulates. In adults, traumata, alcoholism and other such conditions are said to initiate the latent glio-fibrosis. The frequent combination of syringomyelia and primary internal chronic hydrocephalus is said to point to the fact that in both diseases there is merely a varied localization of the congenital gliomatosis. (See also Schrottenbach.) In rickets, a mild acquired hydrocephalus in the first year of life usually undergoes regression. In many cases, however, the etiology remains clothed in darkness (for the older literature, see Weber; see also Weigandt).

The changes in the brain and the skull are less severe than those in congenital hydrocephalus. Ependyma granulations and adhesions between the opposite walls of the ventricles are frequent. If the hydrocephalus begins before the growth of the skull is completed, the skull may acquire enormous dimensions (macrocephalia). Later, this stretching is no longer possible, but its inner layer may be burrowed or excavated or sclerosed as in tumors of the cerebrum, in which the clinical course of increased intracranial pressure, papillo-edema, atrophy of the optic nerve, may be similar to that of hydrocephalus. Occasionally there is rupture into the orbit.

The so-called senile hydrocephalus found almost constantly in very old individuals, is a hydrocephalus ex vacuo (for atrophy, see p. 1863) and, as a rule, of mild degree. According to others, however, it is the consequence of chronic inflammation of the pia-arachnoidea which leads to sub-arachnoideal and ventricular edema and atrophy of the brain as secondary results.

Hemorrhages into the Ventricles

They are quite frequent following rupture of cerebral hemorrhages. The blood is firmly coagulated, occasionally in all ventricles (figs. 932 and 933). Hemorrhage into the pons may rupture into the fourth ventricle, and the latter be filled by a coagulum as big as a plum. Ventricular bleeding may also follow the trauma of extracting the child's head with the obstetrical forceps (see p. 1810, and under trauma). More rarely the blood comes into the ventricles from the base of the brain (rupture of an aneurysm) by way of the subarachnoid space.

Ependymitis

Two distinct entities are understood by this name:

1. The very frequent overgrowths of glia, which occur (*a*) as the so-called ependyma granulations or ependymitis granularis, in the form of a growth of the subepithelial layers of the glia, in which the epithelium may also take part in gland-like overgrowths (Jeremias); they produce transparent sandy nodules up to the size of a hemp seed. For the histological details and literature, see Saltykow. If the finger is rubbed against the internal surface of the ventricle, the sensation is that of a raw surface. The granulations are roughest and most numerous in the lateral ventricles and in the lateral recess of the fourth ventricle. Granulations on opposite sides of the walls may adhere, and lead to diffuse or stringy union of parts of the ventricular surfaces, particularly in the anterior cornu. They are found most frequently in chronic hydrocephalus, in which, according to Margulis, the primary picture is not inflammatory but sclerosis; they are found also in progressive paralysis in all the ventricles, and very often in the atrophy of old age. (*b*) Furthermore, there may appear diffuse flat scleroses and (*c*) networks of white thickening composed of overgrowths of glia. The inner surface of the ventricle feels tough, leathery, and uneven. The author has seen this condition frequently in multiple sclerosis. See Delamare and Merle concerning other forms of chronic ependymitis.

2. There are also found, but more rarely, simple granulation tissue nodules which occasionally contain giant cells (lit. by Tillgren).

Parasites

Echinococci in the ventricles of the brain are rare. Cysticerci in the walls of the ventricles or in the plexus are fairly common. Less common is the occurrence of free cysts. The author found a cysticercus as large as a pea, loose in the fourth ventricle; during life the patient manifested increased intracranial pressure symptoms as soon as he sat or stood erect, probably because there was a valve-like obstruction of the aqueduct of Sylvius. In a second case, (61 year old man), there was marked dilatation of the fourth ventricle. (See lit. by Sato and Marchand, also Sprotte, Verse, Fraenkel, Herzog.) Free cysticerci have been known to cause sudden death through paralysis of the respiratory center (?); see Pforringer, Askanazy, Verse. They occasionally call forth granular, warty, ependyma growths and may adhere to the walls of the ventricles. Some of those free in the ventricle, particularly in the fourth, belong to the form of cysticercus racemosus (lit. in appendix, p. 1845).

For tumors, see page 1838 and following.

DISEASES OF THE BRAIN SUBSTANCE

Malformations of the Brain

(A few of the most important malformations will be discussed. For normal anatomy, see Edinger, Zwölf Vorlesungen uber den Bau der nervosen Zentralorgane, Leipzig. For pathology, see von Monakow, Gehirnpathologie, Wien, 1905. See also Spielmeyer, Histopathologie des Nervensystems, I, Berlin, Springer, 1922.)

Malformations of the cerebrum accompanied by failure of development of the bony parts of the skull.

In **anencephalia,** which is explained by a failure of closure of the embryonal medullary groove following adhesions to the amnion, the brain is entirely absent or present only in traces. Total anencephalia is accompanied frequently by total cleavage of the skull, (cranioschisis totalis), which consists either of an entire lack of the vault of the skull (acrania), or a condition in which only parts (posterior fossa with the foramen magnum) are evolved (hemicrania or hemicephalia). Of brain substance there is practically none, or, more often, in the crooked base of the skull there is a nodular red mass composed of connective tissue and blood vessels and mere traces of specific cerebral substance (acrania or hemicrania with exencephalia); there may be no trace of skin on the skull (fig. 924). The spinal cord, also, is usually poorly developed. As a rule, there is a coexistent micromyelia (Petren, v. Monakow, lit.). If the vertebral column is also open along more or less of its surface (craniorachischisis), the mass lies more toward the neck (notencephalia). (For

further details and the most important literature, see Ernst, Missbildungen des Nervensystems im Handbuch von E. Schwalbe, 1909.) The eyes are developed, but are relatively large and protuberant, while the forehead is receding. The ear is pushed forward (Marx). This peculiar appearance has led to the term anencephalia. The rarer iniencephalus is similar to notencephalus, and is characterized by hydrocephalia and posterior encephalocele, which extends into a fissure of the cervical spinal column; the distended posterior part of the head is pulled backward and thus covers the rachischisis. The scalp blends with the skin of the back

FIG. 923.—Acrania and rachischisis in cervical spinal cord; premature birth; ½ nat. size.

FIG. 924.—Hemicephalia; cranioschisis; hemicrania with exencephaly; ⅔ nat. size.

(Marchand, Hunziker, Michel, lit.). (For the adrenals in anencephalia, see p. 1280.) Occasionally there is also a thymus hyperplasia (author's case, weight 26 grams). The author studied an anencephalus which was the third child of this kind from the same, apparently healthy, parents.

Cephalocele, Hernia Cerebri.—When the skull is partially undeveloped as a result of defects in ossification, and a segment remains unossified (the most frequent form; Siegenbeek van Heukelom), or when there is overstretching and protrusion of parts of the brain following ventricular hydrops in early embryonal life (Exner, lit.), or when an abnormally increased growth of parts of the brain due to malformations of the neural anlage prevents closure of the skull in one situation, or when amniotic bands, adherent to the meninges, pull parts of the brain outward, the

condition is spoken of as fissure of the skull. If, at the same time, there is a defect of the dura (Muscatello, lit.), through which the contents of the skull protrude, the condition is known as hernia cerebralis, or cephalocele. The most frequent situation is median in the occipital region, occipital hernia between the fontanel and the foramen magnum; it may also be lateral, or in the base of the skull, in the region of the sella turcica—in which case the hernia protrudes into the pharynx—or, in the region of the frontal bone near the root of the nose, or below the bones of the nose (Zingerle), or laterally against the orbits, or even in the upper nasal cavities (very rare). The protruding sac in typical cases is covered by skin, and may be composed of different parts; (*a*) in a meningocele cerebralis or a

FIG. 925.—Occipital hydrencephalocele; boy 5 months old; ½ nat. size.

hydromeningocele, the hernial sac is filled with fluid and is composed of scalp and arachnoidea; hair and sweat glands from the skin may grow deep into the arachnoidea (Salzer). (*b*) In an encephalocystocele or a hydrenocephalocele, the sac is composed of changed cerebral substance with the soft membranes; usually the hydropic ventricle also juts forth; the dura is lacking in (*a*) and (*b*). The sac may be much larger than the head itself. (*c*) In an encephalocysto-meningocele or a hydrencephalomeningocele, there is protrusion of the brain substance with a portion of the ventricle into a localized sac composed of arachnoidea and filled with fluid. The dura is lacking. (*d*) In an encephalocele, the normal, or the changed cerebrum, with its membrane, prolapses; there is no fluid; there are often coincident malformations; thus in a case of Ernst, heterotopia and microgyria in the cerebellum (lit. by v. Monakow). Hernias of the cerebellum are rarer, with positions corresponding to the fissures in the occipital bones near to the foramen magnum (Chiari and v. Monakow). The prolapsed parts are predisposed to circulatory disturbances, trauma and infections.

Malformations of the Brain with Closed Skull.—Micrencephalia is a hindrance in development of the brain as a whole (hypoplasia), but it is free of gross defects; the skull is closed, abnormally small (microcephalia), but disproportionate, since the other parts of the skull are small in comparison to the bones of the face (fig. 928). The remainder of the body may be of normal size. But in nannocephalia there is a dwarfed body with a small head. It is not always easy to differentiate in a given case if the condition is due to a primary hypoplasia of the brain, or a secondary hypoplasia due to a disturbance in the growth of the skull (Virchow), *e.g.*, through premature synostosis of the sutures (p. 1188). But because of

FIG. 926.—Fetus with various cerebral hernias and harelip, due to amniotic cords. There was also a tumor of r. hand; bilateral pes varo-equinus; other amniotic cords.

the marked abnormalities of the brain, which is often relatively much smaller than the skull, and because they are not always accompanied by similarly severe changes in the skull, particularly not with premature synostoses, it is quite probable that the condition is primarily a disturbance in the growth of the brain, which is felt secondarily in the skull, so that this structure also remains underdeveloped (Aeby; Pfleger and Pilcz, lit.). There is hardly one case in the literature which unquestionably demonstrates that there was primary premature synostosis as the cause of micrencephalia, even though the skull occasionally, but not always, may have shown early closed sutures. The theory of C. Vogt, based, however, only on examination of skulls, that in micrencephalia there has been an atavistic backward tendency, has been abandoned (H. Vogt, lit.); these brains show only a superficial resemblance to animal forms, such as would be expected by the laws of phylogenetic inheritance. The severity of the

changes in the brain is varied. Single parts may be smaller, particularly the occipital lobe, whereby the usually normal cerebellum is not covered, or the cerebrum as a whole is small and poor in convolutions, although those that are present may be plump (macrogyria), or small (microgyria); or single parts, particularly of the cerebrum, may be only rudiments (extreme cases). In a 3½ weeks old, very large, strong, girl, who, according to the physicians, was quite vigorous, the author found that the entire anterior part of the brain was no larger than a small cherry, while the cerebellum and pons, medulla oblongata and the nerves, as well as the basal vessels, were completely developed; the small cranial cavity was filled almost completely by these parts; there was mild external hydrocephalus. In a case described by Rohon, the brain was quite similar and lay in an exceptionally small scaphocephalic skull (3 weeks old boy). The

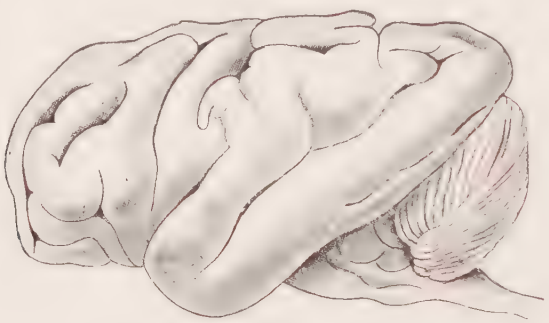


Fig. 927.—Brain of Helene Becker, microcephalic, (after Bischoff); weight of brain 219 gms. (normal 1377 gms.).

brain weighed but 17.3 grams, and is the smallest brain on record. Even the weight of the brain of older microcephalics may be diminished; thus in the 8 year old Helene Becker (von Bischoff), the fresh brain weighed 219 grams, and the brain of a 15 year old girl (Marie) in the same family weighed only 152 grams. Among 9 children of this family, there were 5 microcephalics, born of the same mother, but her husband had 3 normal children by a previous marriage. (For more details concerning this interesting family and literature, see Dannenberger.) The average weight of the brain in male adults is 1360 grams; (according to Weigner 1355 grams, according to Handmann 1370 grams, according to Marchand and Schonfeld 1400 grams); in females 1220 grams; (according to Marchand and Schonfeld 1275 grams); *i.e.*, about 2% of the body weight. The specific gravity of cerebral substance is 1.040. From the capacity of the macerated skull the weight of the brain can be calculated by subtracting 8.5%, and multiplying the result by 1.040 (Marchand). According to Reichardt, all weights in brain pathology are unreliable, unless there is

taken into consideration the capacity of the skull, *i.e.*, the relation of the space in the skull without the dura to the brain weight which under normal conditions is as 100 to 90. The difference between these two figures, normally about 10, is said to account for the differences published in regard to cerebral weight. Cerebral weight in female newborns is 380 grams, in males, 400 grams. The final size is reached by the brain in the seventeenth or eighteenth year. The normal changes of age begin in about the fiftieth year. In a seven year old idiot (typical microcephalic with retreating forehead, arched nose, protruding upper jaw and small lower jaw, broad mouth, diminutive skull), the author found that the

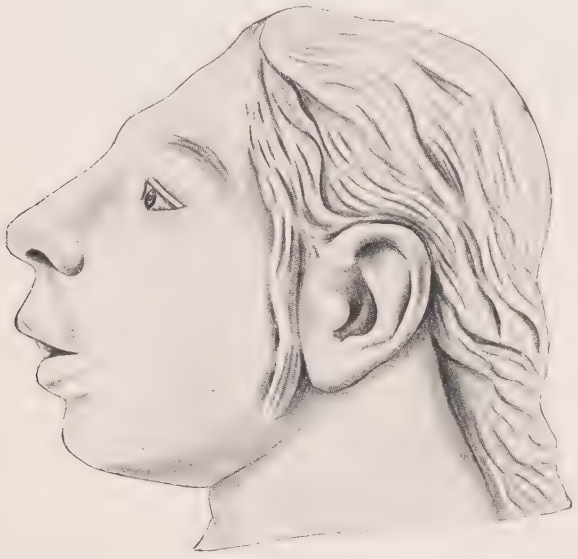


FIG. 928.—Plaster of Paris cast (from life in 7th year) of head of the microcephalic Margarete Becker; small skull, receding forehead; born, Sept. 3, 1870; still alive, 1912. Circumference of head in seventh year, 370 mm.; at age of 42, 401 mm. (see Dannenberger).

brain weighed 650 grams; this case is one of moderate degree of microcephalia (500 to 800 grams, Marchand). There are no definite figures available for the relationship between the brain weight, the body length and the body weight. The histological composition of the cortex is usually described as "incomplete;" it is filled with abnormal cells difficult to recognize; in all probability it contains but few nerve cells. The incomplete formation of the brain is often expressed by heterotopias, particularly from the cortex into the medulla. The causes of microcephalia are unknown (see Marchand). Many assume that it is due to fetal cerebral diseases, as well as to primary defective growth, cases of primary agenetic origin have been called microcephalia vera. (Lit. by Giacomini, Marchand, v. Monakow, Anton, Probst, Hilty, H. Vogt, lit.).

True hypertrophy, or hyperplasia of the brain (macrencephalia with corresponding macrocephalia) is rare; most of the cases described as such are in reality pseudohypertrophies, or enlargements because of pathological processes; thus the cases of Volland (22 year old man, brain weight 1874 grams) and Schmincke (lit.), (35 years old, abnormal psychic, male, brain weight 2155 grams) were demonstrated due to an abnormal hyperplastic development of neuroglia. But there have been cases reported of true, or pure enlargement, and increase of the parenchymatous and interstitial parts (Marburg; hyperplasia of the cerebellum, see Spiegel). v. Hansemann described a true megalencephalia (16 year old boy, brain weight 1860 grams); microscopically there were no pathological changes; he believed it was due to a "faulty" congenital anlage. In a 16 year old, very intelligent boy, the author found a brain weight of 1700 grams. The diagnosis "true macrencephalia" may be made only after careful histological examination. Psychic ability and brain weight are, however, not parallel, and even though very important humans may have heavy brains (Byron, 1807 grams; Cromwell, 2000 grams; Turgenjeff, 2012 grams), deductions are uncertain concerning the intelligence from the size of the skull and the brain (W. Fuchs). v. Walsem who has reported the highest observed weight of the brain, 2850 grams, obtained the specimen from a 21 year old idiot (lit. by Anton, Marchand and Handmann, Fritze, lit.). On the other hand, a decrease in weight below 1000 grams in middle aged males, and under 950 grams after the sixtieth year, is at least suspicious of psychic disease (Mittenzweig, Scharpff, Marchand). But it must be remembered that a number of psychic diseases are accompanied by swelling of the brain (Reichardt, Krueger), which produces an increase in weight. Epileptics who die in the status epilepticus in contrast to normal individuals, often show a significant increase in brain weight (Krueger, lit.). In "constitutional," which means lymphatic, suicides, Miloslavich found an increase in the brain weight (average, 1521½).

Partial hypoplasias are most frequent in the hemispheres of the cerebrum and the cerebellum. See Sternberg concerning complete aplasia of the cerebellum; concerning hypoplasia (secondary developmental inhibition) of the cerebellum, see Dietrich.

Microgyria, particularly prone to occur in the cerebrum, may also appear in the cerebellum, and is differentiated into false and true. In false microgyria, otherwise normal convolutions are wrinkled and hardened, so that they appear smaller and thin and comb-like. This is believed to be due to various pathological sclerosing processes during fetal life or early childhood, such as encephalitis, meningitis, hemorrhages, softening. In true microgyria, on the other hand, there is an increased production of small convolutions (polygyria) which leads to ridges and folds, and an

appearance which has often been compared to that of a cauliflower. The smaller gyri can be recognized only on cross section (internal microgyria, Obersteiner). The cases include porencephalia, in which the edges and bases of the convolutions are microgyric, cases of marked hydrocephalus internus, as well as failures of development of the furrows and other developmental disturbances. In true microgyria, v. Monakow believes the characteristics are a halt in the development of the nerve cells, a perverted grouping, as well as a gliosis, and a scattered heterotopia of gray substance (cortical metaplasia). As an uncomplicated condition, it is rare. There are also combinations of false and true forms. The symptoms of fetal inhibition may be combined with those of inflammatory processes, and it is then difficult to differentiate if the inflammatory processes are primary or secondary (H. Vogt). For more details and lit., see Nieuwenhuijse. Unilateral microgyria produces asymmetry of the brain.

Status verrucosus deformis, (Ranke, lit.), is a condition characterized by the presence of small, tough-walled ridges on the surface of the cortex, a persistence of the "warts of Retzius" beyond the fourth embryonal month. According to Alzheimer, epilepsy is always coincident.

Partial agenesia is likewise seen most frequently in the hemispheres of the cerebrum and the cerebellum. The corpus callosum may be lacking in part or completely (lit. Arndt-Sklarek, Groz and particularly Marchand, Stocker, Landsbergen, lit.), or it may be the fornix and the small commissures. Partial cerebellar defect with total absence of the corpus callosum has been described by the author (hypoplasia of the cerebellum, see Henschen, Dietrich; total aplasia, see C. Sternberg; lit. by H. Vogt and Astwazaturow concerning congenital diseases and malformations of the cerebellum). These may be (*a*) primary developmental disturbances, pure agenesia, or (*b*) a pathological formation of fissures, or of cortical furrows, or (*c*) softening and absorption of parts in earlier times, from inflammations (fetal meningitis, encephalitis), traumata, and circulatory disturbances such as thrombosis, embolism and hemorrhage. In the rare hydranencephalia (Cruveilhier), the skull is normal, the brain is partially or totally defective (following disease of the vessels, Kluge) and the space liberated by the absent brain is filled with hydrocephalic fluid. In an observation of the author, this fluid was cloudy red and contained many fat droplets; the hemispheres of the cerebrum were lacking, the middle parts of the brain were present, the adrenals were markedly hypoplastic, the thymus was large. In hydromicrencephalia the skull is smaller, the fontanels are closed or open, as they were in a 46 cm. long dolichocephalic newborn which lived two days. Collections of

fluid are found in the more or less defective brain, which produce vesicular spaces, communicating with the middle or the lateral ventricles. Where circumscribed areas of the brain were destroyed, there appear cystic defects, furrows, funnel-shaped holes, or wide open deep spaces in the brain substance, which may reach into the ventricles, or, more rarely, from the ventricles out into the white matter and still be covered by cortex. According to Heschl and Kundrat, all of these various defects should be called porencephalias. In the circumscribed varieties there may be differentiated: (*a*) "typical," which occur congenitally, and in which the pores or spaces are usually in the neighborhood of the central parietal lobes or on its borders, thus in the region of the artery of the Sylvian fissure, or in the height of the convexity. They lead to cerebral childhood palsies (hemiplegia spastica infantilis). Around the edges of the defect, which may be bridged by arachnoidea and over which the pia shrinks, the convolutions are swollen, overhanging, and show a radiating appearance converging to the pore. But the pores may also be found occasionally in other parts of the brain. The condition is supposed by many to be due to "developmental anomalies" (Schattenberg, v. Kahlden), thus from abnormal deepening or infolding of the furrows and convolutions in early embryonal life. The condition is also known as congenital primary porencephalia. Hydrocephalus is a frequent accompaniment. The condition may be symmetrical. (*b*) In other cases, the secondary cysts filled with serum are demonstrably due to the fact that parts of the cortex or medulla of the cerebrum or cerebellum have been destroyed or softened, as in figure 935. This occurs most frequently, (Freud), intra- and post partum, or in childhood, from traumata which lead to hemorrhages (see hemorrhages from forceps delivery, page 1810), or directly destroy the brain substance. Porencephalia may also arise through vascular diseases with ischemic necrosis, inflammations, such as primary fetal hydrocephalus, which, as in an observation of Obersteiner, produced a pressure on the ascending branches of the artery of the Sylvian fissure, obstructing the arteries and leading to atrophy of parts of the brain; it may be due to softening, (anemic necrosis), without obstruction of vessels and without inflammation (Kundrat, lit. by Dahlmann, who believes that vessel spasms were responsible for an extensive softening in the brain of a suckling in which there was also the formation of cavities; compare the cases of Hedinger and Meier). Finally, there are all sorts of transitions between the so-called secondary or so-called acquired porencephalias found among adults having penetrating spaces or defects, usually with sclerotic edges, which may be the end results of a destruction, or softening of parts of the brain through traumata, hemorrhages, thrombosis, embolism or encephalitis (fig. 935).

Heschl believed his case was due to fetal meningitis and encephalitis, which led to fatty degeneration of the vessels and to encephalomalacia and resorption of the parts of the brain belonging to these vessels; he found spaces in the cerebrum filled with serum which extended into the ventricles; fatty granular remnants of brain were on the inner surface of the pia. Later the idea gained ground that there was no fundamental difference between the (typical) congenital and the acquired porencephalias. The reason for the latter porencephalia is often easy to recognize, but it is quite difficult in those developing very early, and therefore the explanation, "developmental disturbance," "inhibition of growth," is used, particularly when there is that peculiar radial arrangement of the convolutions around the pore. But now since Beyer has shown beyond question that there really is no fundamental difference in the development of (*a*) or (*b*), and that the same causes which have been demonstrated in (*b*) occur also in (*a*), it is recognized that the peculiarity of the "typical," usually congenital, case lies in the facts that it occurred in fetal life or early youth with a more rapid accumulation of fluid and complete absorption, which, with a lack of fibrous and glious formation, allows the pore to remain. Thus complete topical compensation is brought about by the swollen converging convolutions. As further proof of this theory, viz., that actual destruction of brain substance occurs, we may cite those cases which show obvious unresorbed residues of the destruction. In the latter relation, the author would report a case in a 15 year old girl, in whom, during the last few weeks before death, there were present a hemiplegia and hemianopsia; the pia bulged into the ventricle through a broad cystic porencephalic defect in the middle part of the right hemisphere, while another balloon-like prolongation spread under the floor of the ventricle; in the cystic pial membrane, an intensive brown discoloration composed of granular and crystalline blood pigments pointed to hemorrhage as the cause of the porencephalia. There was a general hydrocephalus, marked (secondary) asymmetry of the skull, particularly of the base; history of trauma occurring some years before. (Lit. by Schutte, Shirres, Anton, Zingerle, J. Kopp; lit. Spatz.)

The various kinds of heterotopia of the gray matter, very frequent in microgyria and in microcephalia, may be mentioned; cortical substance is found where normally it does not exist. Thus, there are gray masses in the middle of the white matter, or in the walls of the ventricle near the basal ganglia, and others. Moreover, metaplasias occur in the form of abnormalities in the arrangement of the various cell complexes, (Meine, Kotschetkowa, v. Monakow, Ernst, H. Vogt, lit.; lit. by Ernst). H. Vogt and Astwazaturow point out that heterotopias occasionally develop in a false direction, and thus are on the border line between malformations and tumors.

Heterotopia of plexus tissue has been described by Kitabayashi in the cerebellum, the hippocampus major, and the geniculate body of a patient with psychic disease.

Regressive Changes (Atrophy, Softening) of the Brain

Atrophy of the Brain.—This may be local or general.

General Atrophy.—The entire brain becomes smaller, inasmuch as the nerve elements—the ganglion cells, but particularly the nerve fibers of the cortex and the white matter—undergo degeneration in the form of simple or parenchymatous atrophy. A generalized simple diminution in size is seen in old age—the atrophy of old age,—involving the tangential fibers of the cortex and particularly the ganglion cells in shrinking, disappearance of the cell granules, increase in pigments, and loss of prolongations. It occurs, furthermore, in diseases of long duration, such as chronic lead poisoning, alcoholism, etc. In the latter, it is not rarely, in old age it is constantly, combined with an increase of the interstitial substance, *i.e.*, increase and hardening of the neuroglia (atrophic induration).

According to Ciarla, the so-called "senile plaques" are found in these cases (compare below under senile dementia). For details concerning the histology and the changes of old age, see Miayake, Siemerling, lit.; Monckeberg, F. H. Lewy. Concerning the senile brain, see Leri, Calmettes.

The meninges are thickened; the vessels show regressive changes, such as are found in senile dementia.

But, according to Simchowicz, the changes in senile dementia are rather in the ganglion cells which show fatty pigmented degeneration, sclerosis with fatty degeneration (foaminess), and finally, granular degeneration to a particularly extensive degree. Furthermore, Simchowicz believes that senile plaques are quite pathognomonic, depending on primary necrobiotic processes in the glious and nervous layers, to which are added reactive processes among the glia cells in the periphery of the plaques (Ciarla). The "senile plaques" are also called "glands" (O. Fischer), because the precipitation of an unknown substance in the degenerated glious and nervous layers produces gland-like pictures which resemble colonies of fungi. The ganglion cells of the middle layers of the cortex in which these "glands" are found, particularly in those cases of presenile dementia showing focal symptoms (Alzheim's disease), may contain tufted or coiled fibers (fibrillar change, Alzheimer), or rough fibrous fibril formation; (for more details see Schonfeld, lit.). Senile dementia is also looked upon anatomically as an increase in the ordinary senile involution changes, particularly those in the cortex.

In paralytic dementia or the paralysis of the insane, the atrophy may be very extensive, but the sclerosis is not ordinarily marked.

See literature by Fritze concerning brain weights.

In general atrophy of the body, starvation, etc., the brain does not lose weight as do other organs (Dunschmann, Krieger, lit.).

The **autopsy findings:** in marked atrophy, the weight of the brain is decreased; the organ is shrunken, does not completely fill the skull; the convolutions are finer, smaller, often sharply angular, comb-like, and far apart; there is much fluid in the subarachnoid spaces, a complementary process, (hydrocephalus externus ex vacuo). The ventricular fluid is increased ("senile hydrocephalus;" see hydrocephalus internus ex vacuo); the ependyma is thick, often granular. The base of the ventricle (thalamus, corpus striatum) is shrunken, wavy, and flattened. On section through the cerebrum, (through the basal ganglia), the substance is retracted from the neighborhood of the vessels, so that the perivascular lymph spaces appear abnormally wide, even cystic, (perivascular brain cysts, "etat crible" according to Durand-Fardel, or sieve-like degeneration), and the tissue is full of holes like a Swiss cheese. (A much more outstanding type of the so-called Swiss cheese brain—porose cerebrale, P. Marie—occurs as a post mortem change when gas producing bacteria are present ante-mortem in the blood and have invaded the brain; see p. 1755; lit. by Chiari.) There is stasis of lymph from thickening of the pia-arachnoidea (Borst), since the so-called perivascular lymph spaces empty their contents into the subarachnoid space. Gliosis is observed around the cyst, or a fibrous reticulum is found, even to complete cicatrization (Saltykow). The anatomical picture produced by arteriosclerosis is entirely different, for in the dementia of arteriosclerosis, Krueger found the weight of the brain about normal. See the review by A. Jakob concerning the histopathology of the central nervous system in psychic diseases.

In so-called **Wilson's disease** (progressive lenticular degeneration) there is a familial tendency, but, according to the belief of most authors, with which, however, Hall (lit.) disagrees, it is not hereditary, but occurs in younger individuals in whom there is electively a symmetrical degeneration of the cells and fibers of the lenticular nucleus, (the putamen, the outer, and particularly the globus pallidus, the inner portion of the nucleus lentiformis), and in whom there are also glious fat droplet cells, and glia and blood vessel increases. But the changes also appear in the cortex of the brain and in the dentate nucleus (L'Hermitte, Spielmeyer). It is combined with a change in the liver, which, however, does not usually give clinical liver symptoms such as ascites and icterus. This has been mentioned on pages 970 and 971 (see fig. 470); see there, for the opinions

concerning the etiology and the correlation of these localizations. According to Hall, the changes in the liver and in the brain run parallel; both are said to be due to a decreased resistance of the organs (endogenous factor), while exogenous factors at the most produce only an initiative stimulus. Wilson related the symptom complex named after him to paralysis agitans (see also Dziembowski, lit.). The clinical symptoms usually appear slowly; as a rule, the first disturbance is in the motion of the arms, with rhythmic tremors, general muscular stiffness (hypertony) appearing then in the muscles of deglutition and speech, producing dysphagia and dysarthria; this is combined with a certain amount of paresis of the muscles, but not actual paralysis. Psychic symptoms such as irritability, poor judgment, dementia, are often present. The duration of the disease is months or years. According to H. Oppenheim, Strumpell (lit.), Hall, and others, Strumpell-Westphal's pseudosclerosis belongs to the same group as Wilson's disease. They are merely two dissimilar types. The changes in the cortex of the brain, the basal ganglia and lenticular nucleus, with liver involvement and enlargement of the spleen, are common to both, but the lenticular degeneration dominates Wilson's disease (see also Rausch and Schilder). Alzheimer found giant glia cells and changes in the size, form and chromatin content of the glia cell nuclei (Westphal, Spielmeyer). Some cases have a peculiar symptom in the form of a brownish green pigment ring in the outer zone of the cornea (Kayser, Fleischer, Strumpell, H. Oppenheim, v. Dziembowski and others). According to Strumpell, in contrast to Wilson's disease, the stiffness is marked only in the muscles of the face (mimic rigidity). For the histological details of both diseases, see Spielmeyer, lit. [For description of cases in a family and excellent discussion, see Barnes, Stanley, and Hurst, E. W., Brain, 48: 279: 1925; 49: 36: 1926; and 49: 36: 1926.—*Translator.*]

Progressive paralysis of the insane, or dementia paralytica, or simply, paralysis; popular designation—softening of the brain.

This disease is much more common in men, occurring, usually, between the thirtieth and fiftieth years. Syphilis is the determining etiology. The time between infection and the appearance of paralysis varies considerably, as a rule, between five and twenty years, but according to Berger, between ten and twelve years. Sometimes the syphilis ran a previous, almost symptomless, course. Formerly, paralysis, as well as tabes, were termed "metasyphilitic" processes, but since Noguchi in 1913 both demonstrated the presence of the spirocheta pallida in the brains of paralytics and reproduced the disease in animals, (Jakob, Plaut), it is recognized that it is a particular form of syphilis of the central nervous system, in which the histological changes are due to the presence of the spirochetae.

Numerous recent investigations, (see also p. 1868), have shown that the spirochetae exhibit a predilection for the cortex of the anterior parts of the brain, and produce either a localized process in sharply outlined foci (Hauptmann), or a diffuse process, and that the paralytic attacks are related to a sudden and massive destruction of nervous tissue, representing an extensive increase in the spirochetae and their activities (Jakob, Jahnel; see also Levaditi-Marie-Bankowski and Levaditi and Marie, Verse, lit. and others).

According to Jahnel, the best method of demonstrating the spirochetae in the brain is with pyridin-uranium after hardening in 10% formalin; previously, small pieces of the cortical substance were taken, rubbed up in sodium chloride solution and examined under the dark field microscope directly for spirochetae.

Naturally, the demonstration of spirochetae did not answer all of the questions presented by paralysis (Sioli). Other etiological assistance has been postulated. When exogenous injuries such as traumata, overexertion, alcoholism, were shown to have no certain relation (Bonhoeffer), some authors assumed that a congenital abnormal anlage, which produced an abnormal bas relief of the cerebral hemispheres, led to a decreased resistance of the brain ab ova, and thus assured a necessary predisposing cause for the development of the paralytic cortical process (Nacke, Ranke and others); quite recently, Binswanger and Schaxel have stated that in younger paralytics (third and fourth decade), there are insufficient cerebral vessels as constitutional factors allowing the development of the spirochetae in the brain. Undoubtedly other factors are also important; thus, it is known that paralysis is much more frequent in intellectual workers than in individuals who work with their muscles; therefore, paralysis has been viewed from the standpoint of the "over exertion diseases" of Edinger; it has been demonstrated that increase in the culture of a community brings forth an increased amount of paralysis (Krapelin, W. Gartner, lit.). Saturation with syphilis alone is therefore not completely determining. The relatively large percentage occurring among Jews is also well known (see, for example, A. Cramer).

For the clinical diagnosis of paralysis the following examinations have long proved useful: of the blood serum; of the cerebrospinal fluid; in order to demonstrate the syphilitic antibodies in the blood serum, (Wassermann, Citron, Nonne and others); also increased lymphocyte content of the cerebrospinal fluid. On the basis of these examinations Oppenheim, Nonne and others arrived at the opinion that the great majority of paralytics were also syphilitics. In 156 paralytics, Plaut found the Wassermann reaction of the blood serum uniformly positive (if the serum was negative, paralysis in all probability could be excluded); in 147 spinal

fluids coming from certain paralytics, only six were negative (compare the results with those of tabes). It must be remembered, however, that a positive serum reaction indicates with a great deal of probability that the individual has, or has had, lues, but not that the condition under discussion is syphilitic, since there are cases of syphilis, not only of the brain and spinal cord, but also of other situations, in which the serum is negative (Oppenheim and others). The paralysis is characterized clinically by a progressive diminution in intelligence, disturbances of speech, particularly

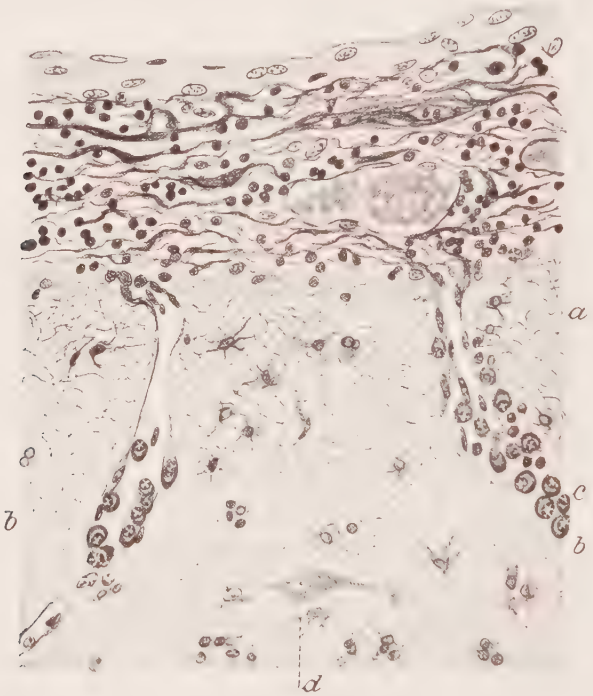


FIG. 929.—Tender meninges and cortex in progressive paralysis; cellular infiltration (lymphocytic) of the fibrous thickened pia-arachnoid; two capillaries, passing into cortex, are surrounded by plasma cells (c) and isolated lymphocytes. Outer layer, poor in cells a, contains glia cells and fibres. The cellular layer b shows but one ganglion cell (d); the rest disappeared; only a delicate glia network and glia cells remain. Iron hematoxylin eosin stain. (High power.)

elision of syllables, paralytic attacks of apoplectiform or epileptiform character, and reflex fixation of the pupils which usually begins early—the pupils remain quite small. Paralysis is incurable with very few exceptions (see Nonne), is progressive, and ends in months or a few years with death. It attacks the brain chiefly, but the spinal cord is also involved to a certain degree. The latter shows both primary tract degenerations and secondary descending degenerations from the cortical foci. There is degeneration of the pyramidal tracts in the lateral and anterior, or the posterior bundles, with certain differentiations from tabes in

both the localization and time intervals; but both diseases may be combined, which is quite frequent, (compare Westphal). Since the early paralyses tend to hide the ataxia, the typical picture of tabes may be difficult to find. There is always a chronic inflammatory spinal meningitis, and always infiltrates around the blood vessels, particularly their adventitiae.

The rare juvenile paralysis is more common in women; for this, lues, usually congenital, is likewise responsible. [For case reports, see Klauder, J. V. and Solomon, H. C., Am. J. Med. Sci., 166:545:1923.—*Translator.*]

In its macroscopic aspect the brain of paralysis is similar to that of atrophy as described on p. 1863. But it must be remembered that the soft meninges are frequently thickened and cloudy, and because they are adherent to the cortex of the brain, they can be removed only by taking along with them portions of the cortex; furthermore, the atrophy is more marked in the cerebrum, chiefly in the frontal and parietal lobes; the gray matter of the cortex may be much diminished, so that its weight is reduced one-third to one-fourth, while the weight of the brain as a whole may sink, in the male, to 1000 grams. Occasionally there is a coexisting internal hemorrhagic pachymeningitis. The pathological process consists, first, of a slow, progressive disappearance of the nervous elements in the cortical substance of the cerebral hemispheres (Wernicke), and, secondly, of chronic inflammatory processes in the blood vascular apparatus.

The disappearance is chiefly of the finer medullated nerve fibers, and the tangential fibers in the upper layers of the cortex, particularly those in the gyrus rectus and in the island of Reil. Newer methods of staining (neurofibrillar stains of Bielschowsky) have shown, however, that many nerve fibers are present which are not shown with the other medullated nerve stains (Moriyasu). The ganglion cells of the cortex are often degenerated, sparse in nuclei, vacuolated, pigmented, atrophic and fatty, (for more details, see Nissl). Morijasu demonstrated the destruction of the ganglion cells quite clearly; there is disappearance of the prolongations as well as of the fine intracellular fibrillae, with later destruction of the extracellular fibrillae. These degenerative changes are combined with characteristic diffuse infiltrates consisting of lymphocytes and plasma cells (Alzheimer; coming from adventitiae, see Roth), as well as with the production of yellow, hemosiderin-containing pigment granules in the lattice cells of the meninges and adventitial spaces (compare also Lubarsch, Spatz). Infiltrates are particularly thick in the cortex, to less degree in the white matter; they are most marked in the central and frontal convolutions, but are also found in the spinal cord (Weiss). The pia mater is often richly infiltrated and thickened; the collections are

partly diffuse, and partly along the vessels in the cortex (meningo-encephalitis); adhesion of the meninges to the brain surface is common (chronic adhesive leptomeningitis). The supporting tissue of the brain shows increase in both the cells and fibers of the glia (Spielmeyer), probably as a secondary reaction to the loss of tissue, but also, in part, at least, as an active growth. At the same time, there are degeneration and destruction of the nuclei and the bodies of the glia cells (Weber). But Schaffer believes the changes in the glia are independent, the glia, as well as the nervous parts, is diseased, and the glia overgrowth is always significant. According to Bratz, 25% of the cases show sclerosis of the hippocampus major, which is also found, among other conditions, in idiocy, imbecility, genuine epilepsy in 50% of the cases. Furthermore, there are frequent thickenings, hyalin changes and adventitial infiltrations (plasma cells) of the smaller arteries and capillaries, also overgrowth of the intima in the smallest vessels with the formation of new vessels through budding. In contrast to the findings in ordinary syphilis, which usually responds to therapy, Noguchi seldom found the spirochetae in the neighborhood of blood vessels and practically never in the vessel walls of the cortex. Jahnel demonstrated them in the perivascular spaces of the white matter, and Hauptmann found that the vessels may be infiltrated by spirochetae without showing local changes, particularly without endarteritis. The degenerative changes are believed by some to be directly due to the activity of the spirochetae (Verse), but by others to be dependent on vessel changes; they occur in addition to the inflammatory changes (see Jahnel). In the cortex, Hauptmann, Schob and others demonstrated both miliary necroses surrounded by spirochetae and infiltrated by leucocytes, and miliary abscesses containing spirochetae. An earlier opinion (accepted by Wernicke, Tuczek, Strumpell, Alzheimer and others) stated that degeneration of the nervous elements was the primary condition, and infiltration and growth processes in the blood vascular apparatus were secondary; others postulated an interstitial inflammatory process coming from the vessels and the neuroglia (interstitial encephalitis, Mendel). Plaut believed the inflammatory process in the pia was syphilitic (not metasyphilitic), and was present years before the appearance of the paralysis. But, according to later opinions, the spirochetae are responsible, not only for the histological conditions of the ectodermal, but also of the mesodermal parts of the brain (Raecke). It is interesting to remember that inflammatory foci with a few miliary gummata are common in the cerebrum, particularly when paralyses occur in attacks; there is, however, no definite specific reaction of the tissue as in a typical gummatous encephalitis, but rather an ordinary diffuse inflammation wherein the infectious process manifests a malignant character (Jakob).

Plasma cell infiltrates, as an expression of a chronic inflammatory process, are found in the pia, in which, however, Noguchi could demonstrate no spirochetae; the same cells are found in the adventitial spaces of the vessels of the spinal cord (Oppenheim, E. Meyer, Weiss, Roth, lit.).

An atypical form of progressive paralysis of long duration has been described by Jakob as a syphilitic endarteritis of the small cortical cerebral vessels.

It is worthy of note that the similarities of the changes in the central nervous system in sleeping sickness (p. 211) and paralysis are quite extensive (Spielmeyer, Spielmeyer and Apelt), a fact which has been used as a proof of Schaudinn's belief that the spirochetae pallidae and the trypanosoma are closely related.

Partial atrophy of the brain substance (focal degeneration) appears from compression of tumors, inflammations, from simple circulatory disturbances, which are independent of vessel changes, *e.g.*, in the neighborhood of hemorrhagic and softened foci. Thus, after destruction of the optic thalamus and corpus striatum through hemorrhage, the entire hemisphere undergoes atrophy. Many atrophies involve entire systems in the brain and spinal cord, and produce secondary degenerations, which appear after interruption of the communications between the nerve fibers and their corresponding nerve cells.

This is occasionally seen in the cerebral paralysis of children (hemiplegia spastica infantilis). This disease consists chiefly of paralysis of the extremities of one side, present at birth, or appearing shortly after birth, *e.g.*, after difficult labor, or appearing suddenly in the first year of life after an infectious disease, such as measles, scarlet fever, etc.; it is often an expression of an acute non-suppurative encephalitis of the motor parts of the brain (Strumpell). If such a case is examined years after its occurrence, there is found, *e.g.*, when the left side was paralyzed, marked atrophy of the cerebral convolutions in the motor region, so that the condition has even been spoken of as porencephalia. Peripherally, the motor pyramidal paths are degenerated throughout their entire length (see under spinal cord; descending degeneration), and the parts are diminished in size. The anatomical picture of the cerebral paralysis of children is otherwise not uniform, and etiologically there occur traumatic as well as infectious or embolic causes; the focus, usually in the cerebrum, may be caused by disease of the blood vessels with hemorrhages, subdural or cerebral cysts, defects (porencephalia), focal softening, atrophic scleroses, etc. Many cases are the result of endogenous germinal defects, and show anencephalia, partial aplasias, microgyria of an entire hemisphere; more rarely, there is an increase in volume of various parts (Lindemann and von Marenholtz, Durck). Diminution in intelligence and

epilepsy are often manifested during the course of the cerebral paralysis of children.

A secondary atrophy in the centripetal direction (retrograde degeneration, see p. 1873), is seen after enucleation of the eye ball in younger individuals, as determined by v. Guden in animal experiments; the atrophy extends along the optic tract, *i.e.*, along the optic nerve and the chiasm, which may appear entirely asymmetric; then, since the fibers in the chiasm pass to the other side, the atrophy becomes symmetric in the optic tract, the pulvinar of the optic thalamus, the lateral geniculate body, the anterior corpora quadrigemina and from these primary optic centers to Gratiolet's optic radiation, through the white matter of the occipital lobe to the cortex of that lobe. The latter is the psychic optic center, which both receives optic stimuli, and reproduces memory pictures of optic relationships. If those processes which make possible the memory of seeing, are held in abeyance, so that circumstances are seen but not recognized, there occurs mind-blindness, which is usually due to bilateral disease of the occipital part of the brain. The reverse occurs after destruction of an occipital lobe, namely, atrophy of the corresponding optic tracts.

The Histological Changes in the Various Tissues Following Destruction of the Brain and Spinal Cord

For more details, see Spielmeyer.

Changes in the Ganglion Cells.—There may be mentioned: (1) swelling of the bodies, prolongations and nuclei; the tigroid (p. 1872) may be retained, or (2) it becomes finely granular.

It occurs also in infectious diseases, intoxications, burns and other conditions.

In various diseases of the ganglion cells, thus in (1) and (3) there appear vacuoles and vesicles which must be distinguished from post-mortem changes or artefacts produced during hardening. (3) Destruction in laminated particles, the so-called granular destruction into rounded bodies and rings, a process of solution called by Nissl "severe cell change," is found, among other conditions, in severe psychoses, intoxications and infections. The same changes are seen in acute destruction of cerebral parts following crushing, anemic or hemorrhagic softenings, in the neighborhood of inflammatory foci (fig. 930-gG).

(4) Fatty, foamy degeneration, (fig. 930-fG), occurs with swelling and granular degeneration under the same circumstances as the latter, particularly in marasmus, general infections, intoxications, and also in certain diseases of the mind (dementia precox).

Muhlmann found a large amount of lipoid material in typhoid fever, analogous to the fat metamorphosis in parenchymatous organs. According to Muhlmann and Krause, fatty and pigment degeneration (see below) cannot be sharply subdivided. See Rachmanow concerning the physiological lipoids of the nervous system.

(5) Calcification of single, or groups of dead ganglion cells takes place after true concussion of the brain without hemorrhagic softening, in arteriosclerotic foci, in scars around older abscesses, and in chronic nutritional disturbances which lead to death of ganglion cells (fig. 930-vGN). Sclerotic nerve cells may also undergo calcification. The cells are thickly encrusted with granules and flakes. Friedlander found they had appeared thirteen days after traumatic softening (lit. by M. B. Schmidt).

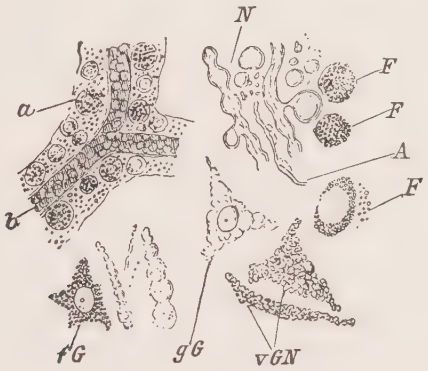


FIG. 930.—a, In the adventitial lymph sheath of the blood vessel (b) lie round cells, fat granule-globules, free fat granules, pigment granule cells; from the periphery of an area of cerebral softening. vGN, ganglion cell and nerve, both calcified; gG, swollen ganglion cell; to the left axis cylinder, swollen and undergoing fragmentation and granular disintegration; fG, fatty degenerated, disintegrating ganglion cell; N, nerves with swollen medulla, partly free myelin droplets; A, axis cylinder almost denuded, shows only traces of attached medulla; from an area of softening 4 days old. F, fat granule globules and fat granule cells. (High power.)

The discovery of Düring is quite important, namely, that the ganglion cells immediately after a trauma, even in the form of a contrecoup (p. 1943), and the cells in the neighborhood of a direct traumatic softening lose their ability to carry out the oxydase reaction (p. 196; Katsunama), which means that there is a disturbance of their function, and loss of the activity of their ferments, representing an initial change in the ganglion cells without change in cell structure. In addition to mechanical insults, those due to toxins and other factors, such as diminished nutrition, lead to the same functional effects.

(6) Simple shrinking may result finally in complete disappearance of cells and their nuclei. It is often accompanied by a sort of condensation of the cells called "pyknosis," in which the cells stain intensively with

many dyes. It is very frequent in chronic diseases of the mind, but may also be found in those with normal minds. (7) The term "sclerosis" describes the result of (6)—a homogeneous waxy, occasionally fissured, condition of the cells; (8) in pigment atrophy or degeneration, the cells, which are smaller, are markedly granular and pigmented brown with "dust" of the Nissl's bodies (see Krause). This is found in psychoses as well as in normals, particularly as a degenerative condition of old age (Muhlmann). The tigroid disappears where the brown granules are present. These pigments have the ability to reduce osmic acid similar to fat.

The expression "fatty pigmented degeneration" is also used. According to Muhlmann, fatty pigment granulation goes through two phases, one, changing of the liposomes, and second, the staining of them; both are catabolic products of the cells. Muhlmann calls the pigment lipochrome; Hueck calls it lipofuscin, and believes it comes secondarily from fat; Lubarsch speaks of catabolic pigment. (See Jonnescu, Oberndorfer, for more details concerning nerve cell pigments.)

Through the use of Bielschowsky's silver method in tissues fixed in formalin, neurofibrillae may be recognized as very fine fibrils, which singly, or in bundles, not only pass through the body of the nerve cells as do the dendrites, but also enter the neurites. See Bickel concerning changes in these under pathological conditions (softening and others). Literature concerning other finer changes of the disintegrating ganglion cells, on p. 1867 and by Spielmeyer, and further by Getzowa, who described the changes in human tetanus.

Neuronophagia is, according to Spielmeyer, the penetration of glious elements into a diseased ganglion cell whose place they usurp, and the destroyed material of which they take up. But leucocytes also become neurono- or necrophages (see under Heine-Medin's disease). Compare pages 1900 and 1919.

Nissl's method of staining ganglion cells (fixation in alcohol, corrosive sublimate, formalin, etc., staining with methylene blue, and neutral red, etc.) brings out, in addition to the nuclei, certain substances in the protoplasm of cells taking the basic dyes; they are in the form of dark granules and waves—Nissl's bodies or granules, and with the exception of the prolongation of the axis cylinder, stretch into the other cell prolongations. Because of the tigroid appearance displayed by the cell after such treatment, v. Lenhossek has given the name tigroid to this substance. After hardening in Müller's fluid, which was formerly much used, the body of the ganglion cell appears melted. The arrangement of these bodies varies in the different cell forms; in the motor cells they are in parallel stripes, and in the spinal ganglion cells the rounded granules leave free the nucleus

and the border zone of the cell. According to A. Bethe, the primitive fibrillae pass through the ganglion cell, and their frayed ends and bundles fill the spaces between Nissl's bodies. Thus the picture of Nissl's bodies is, so to speak, the negative of the fibrillar picture (v. Lenhossek). See Unna and Gans, Held, for the more detailed histochemical structure of Nissl's bodies; the tigroid substance is said to be a nucleoprotein (Spielmeyer).

Under different conditions the tigroid substance is altered; it may appear to change position, to disintegrate, dissolve, completely disappear (tigrolysis, Schmaus), with eventual disappearance of the cells themselves. Among the conditions in which these changes have been observed are poisoning with arsenic, lead, phosphorus, antimony, mercury (Brauer), strychnine, alcohol, the venom of poisonous snakes, prussic acid (Edelmann) and many others (lit. by van Gehuchten); in bacterial intoxications as with tetanus toxin; in changes in the spinal cord (Getzowa, lit.); furthermore, in anemia, inanition, fever, etc. (It is remarkable that the mature ganglion cells of the brains of animals are said to be extraordinarily resistant to the x-ray, Brunner, lit.) Later, the nuclei and the nuclear granules may also change, become homogeneous, shrunken, (pyknosis), etc. Whether Nissl's bodies are nutritive substances, or sources of energy for the nerve cells is still questionable; in fact, their importance, as well as their changes, are still in doubt. According to Held, they can not be seen in entirely fresh cells. Through experimental irritations, changes have been obtained, which afterward were lost (Hodge, Nissl); thus in the paralysis obtained through lumbar anesthesia with stovain (Wossidlo). The possibility of restitution of this tigroid after poisoning has also been demonstrated. It is therefore not quite correct to speak of this condition as "degeneration," and, on the whole, great care is necessary in judging the pathological dignity; this is emphasized particularly by v. Monakow and Oppenheim, as well as by Mourre, the latter of whom points out that in poisoning with the same substance, very different changes may be wrought.

But Nissl's method is useful, among other things, as a sensitive reagent to show the ganglion cells in the so-called retrograde degeneration or retrograde atrophy, the change which is found in the central stump as well as in the nuclei very early (even after 24 hours) after section of peripheral motor or sensory nerves. After a stage of destruction of the granules and an excentric position of the nucleus, there occurs either a regression of the changes—when regeneration takes place in the periphery, or an increase in the changes leading to atrophy of the cells. Retrograde degeneration as such has been known for many years (lit. Durante); after early childhood diseases, in experiments with young animals (Gudden),

and long after amputations, mild changes have been found in the ganglion cells belonging to these parts, but this sensitive and rapid reaction in the entire neuron has only been demonstrated through the use of newer methods, among them, Marchi's fat stain. The retrograde degeneration, which, as a matter of fact, does not always occur in experiments, has been the subject of many interpretations; it is in part an ascending neuritis, and in part a true secondary atrophy, or one which is brought about through damage to the central cells (Lugaro).

Changes in the Nerve Fibers (fig. 930N). When nerve fibers are destroyed by any cause, (crushing, ischemia, intoxication or secondary degeneration), the medullary substance swells irregularly in the axis cylinder, and thus there is produced a "varicose" nerve fiber. Then the fiber breaks into irregular particles, or rounded, or oval, doubly refractile droplets or balls of coagulated myelin (myelin droplets), to be finally destroyed to still finer droplets which give Marchi's fat reaction (fatty degeneration). The axis cylinders either swell focally, and become varicose, or uniformly and markedly (fig. 930 to right of fG); then they break into little pieces and are liquefied to a fluid which accumulates during the destruction of the nerve. But frequently the process is simple atrophy of the nerve fiber, in which the medullary substance slowly disappears and becomes granular, while the axis cylinder is retained for a long time (see multiple sclerosis).

(Marchi's method is performed as follows: pieces of healthy or diseased nerve tissue are placed directly in osmic acid, whereupon the myelin of the medullary sheaths is stained black. If, however, the piece of tissue is first placed for a few days in bichromate of potassium solution or in a solution of formalin, and then in osmic acid for 8 days, the medulla of the nerves becomes black where the myelin has decomposed into glycerophosphoric acid, cholin and stearic acid; the latter is stained black. Where the medulla is normal or where it is totally degenerated, there is no black staining (fat), since no stearic acid is present. Other fats which happen to be present in the preparation also become black, just as in the preparation which is placed directly into osmic acid. Better results are obtained, according to Matuszewski (Schmaus), by using Nissl's method; see in the chapter "nervous tissue" in the "Enzyklopadie der mikrosk. Technik," Berlin-Wien, 1903. See also Spielmeyer, "Technik der mikroskop. Untersuchung des Nervensystems," Berlin, 1911.)

In all places where nervous substance is destroyed, whether by inflammatory degeneration or by non-inflammatory processes, there appear cells which take up the greater part of the products of tissue destruction. These are "fat droplet cells" when they take up fat debris or myelin droplets; "pigment granule cells" when they take up the products of

destruction of blood, and occasionally even entire red blood corpuscles. Some of the granules of destruction do not appear in cells but lie free in the substance of the tissues. In acute destruction of nervous substance, these droplet cells are very numerous, even in a few days, indeed, as the author saw, in 13 hours. In chronic degeneration, chiefly primary, *e.g.*, in tabes, they are fewer in number.

Their phagocytic character is unquestioned; according to Stroebe, they are polynuclear leucocytes in the first days; some, hoewever, are of doubtful origin; according to Stroebe and Hoche, they arise in connective tissue, while, according to F. Marchand, they are adventitial cells of the vessels (Ranvier's clasmatocytes), *i.e.*, of mesodermal origin (lit. concerning genesis of these cells by Schmaus). Fritz Marchand (lit.), on the other hand, believes that the role of the leucocytes in the absorption of products of destruction in the central nervous system is subordinate, and the author must agree with him on the basis of the oxydase reaction in foci of white softening and the multiple foci of sclerosis; in degenerative destruction of the medullary sheaths, it is said that free and mobile glia (ectodermal) cells become droplet cells by phagocyting the products of destruction, while in gross damage of the entire tissue (trauma), cells from the connective tissue also probably act as phagocytes. Merzbacher believes that normal glia cells can become "scavengers," and change to droplet cells. In experimental investigations such as those by Tanaka on rabbits in which small aseptic penetrating wounds were produced by pointed glass rods, the dominant role of the glia in the formation of droplet cells was demonstrated, while the leucocytes, lymphocytes, adventitial cells and fixed connective tissue cells were few in number. For more details, see Ciaccio. In addition to droplet cells, there is another mechanism of removal of products of destruction which takes place when the destructive processes produce smaller particles; this is by means of the amoeboid glia; see p. 1877. For neuronophagia, see p. 1872.

In the neighborhood of recent foci of softening, the fat droplet cells and masses of free fat droplets can be found in the adventitial lymph spaces of the cerebral vessels (fig. 930); thus a large amount of the fluid and granular debris as well as the phagocytic cells are transported from the neighborhood through the lymph channels. Later, these fat granules are destroyed within the cells.

Pigment granule cells may be preserved for long times; a rust brown discoloration appears when they are collected in numbers in the perivascular lymph spaces or in the soft meninges; finally they disappear, or, merely the pigments disappear from within the cells. They are often found for many years around an old scar.

Changes in the Vascular Interstitial Substance

Consequences of Degeneration, and of Foci of Softening.—In many types of degeneration, but particularly in the simple forms, merely the nervous parts undergo destruction. In all foci of softening, however, the glia also is destroyed, but the blood vessels remain, accompanied by their connective tissue; in the spinal cord, the larger strands of connective tissue form a network enclosing the remnants of the soft tissue. But under certain conditions, such as suppuration and ichorous softening, the blood vessels themselves suffer destruction. If the nervous parts alone are destroyed (which usually takes a long time), the remaining homogeneous glia and the blood vessels produce a fine network filled with serum, remnants of the nervous substances and fat droplets. A reparative secondary overgrowth of glia is usually begun and results in a soft or a tough scar. Most of the tissue produced by this secondary overgrowth of glia, in addition to staining by van Gieson's method (the glia is yellow), takes C. Weigert's stain for neuroglia (true neuroglia fiber staining). By this latter method the fibers and nuclei of the glia are stained blue, while the poorly developed cell bodies take no dye. According to Weigert's view, and others, it is shown by this means that—in adults—the neuroglia fibers are connected with the neuroglia cells only per contiguitatem. The fibers do not radiate from all sides as prolongations of the cells, as might be supposed from the stellate figures called Deiter's cells, neuroglia cells, astrocytes, spindle cells, etc., brought into view by Golgi's silver impregnation method; this hides the cell bodies and the fibers penetrating them by precipitates around the nuclei. These figures are not an indivisible morphological complex, but the bright vesicular nucleus, *i.e.*, the neuroglia cell, is merely a middle point, about which the neuroglia fibers are grouped liked an astrocyte, but from which they are entirely different and independent; they must not be thought of as nervous intercellular substance; they merely touch the cells, or, according to Schaffer, dip into the small cell bodies from which they may be differentiated by applying to chrome salt fixed material, borax carmine, indigo carmine, whereupon the nuclei stain red, the protoplasm rose, and the fibers bright green. Other smaller, darker nuclei or glia cells are present, but not in a characteristic position in relation to the fibers. It must be mentioned that this conception of the normal anatomy has been vigorously opposed by Golgi, v. Kolliker, v. Lenhossek, among others, who believe all fibers are prolongations of cells; Streeter agrees with Weigert's conception. According to Held, the neuroglia is a syncytium with many nuclear centers, composed in part of the protoplasmic cell outrunners, and in part of a modification of the cell protoplasm called glia-reticulum, the largest part of which consists of intraprotoplasmic fibers. The

protoplasmic glia functions as a predecessor of the fibers and as a system of meshes, which contains the nervous tissues. Furthermore, according to Schaffer, the glia is not only a supporting apparatus, but also a medium of transfer between the circulation and the ganglion cells. This conception, which negates the teaching of Weigert concerning the independence of the cells from the fibers is shared by the newer work of Kruckmann, Stumpf and others who place the emphasis on the protoplasm. The method of Ramon y Cajal gives exquisite pictures of the glia cells by means of gold solutions in frozen sections fixed in formalin.

At the dense places, which appear gray red or gray (without medullary sheaths), hard, dry, homogeneous, transparent and, to which the term sclerosis, or gliosis is applied (Weigert), there is a thick skein of fibers from the overgrown neuroglia. Other gray places are softer or colloid-like. When, however, as in a focus of softening, both the glia and the nervous parenchyma are destroyed (relatively short time), the defect may be filled either by granulation and connective tissue formation, or, according to Weigert and others (lit. E. Muller), by a vigorous growth of glia which later leads to solid cicatricial masses. The former is more likely to occur in severe lesions.

The connective tissue growth begins in the walls of the vessels and their sheaths; from a richly vascular granulation tissue containing many epithelioid cells (fibroblasts; page 9), there results a hard connective tissue. If the focus is small, and the products of destruction continually absorbed, the defect may be completely filled by the growing glia, which gradually becomes a tough scar as it loses its vascularity from within outward. Large defects, on the other hand, *e.g.*, from hemorrhages or suppuration, may not be filled in because the connective tissue production, or even the overgrowth of glia, is halted at the periphery. Thus, a connective tissue or a glious capsule surrounds a space, or cyst, the walls of which are thick, tough and resisting on the outside, and softer and juicier toward the lumen; the contents are the products of destruction, which are finally absorbed and replaced by serous fluid.

Ameboid Glia.—Following certain acute diseases of the brain which run a fairly stormy course and produce numbers of small scattered foci of destruction, Alzheimer found a pathological increase in the glia, or a modified glia, characterized by small dark nuclei in a cell body showing pseudopodia-like prolongations which contain the characteristically staining products of destruction as carried by ordinary granule cells and by failure of the formation of fibers. Alzheimer, Jakob, Wohlwill (lit.), among others, found these structures in dementia precox, status epilepticus, genuine epilepsy, chronic progressive chorea, intoxications, severe uremia, death from salvarsan, pseudotumor cerebri, as additions

to granule cells in the neighborhood of tumors, in the cortex of early softening and abscesses, in infectious diseases such as scarlet fever, measles and others accompanied by convulsions, as well as in the cases in which a marked serous saturation occurred following circulatory disturbances, such as sinus thrombosis and tuberculous meningitis. This type of decomposition by ameboid glia is also found in tabes (Faworsky). The question of the post mortem formation of ameboid glia, (Eisath, Rosenthal), is believed by Wohlwill to have little importance in pathological findings.

A particular form of overgrowth of the neuroglia is the bushy form in the cerebellum (Spielmeyer, Sittig); the shrubbery consists of spindle-shaped glia cells which form bushes and bands of tissue. According to Sittig, these foci are related in their arrangement to the dendrites of diseased Purkinje's ganglion cells; the band shaped collections represent destroyed dendrites. This glia cell increase may later lead to an increase in the glia fibers. The condition has been found in epileptics, in status epilepticus, paralysis, spotted fever, typhoid fever (Wohlwill, lit.), and in malaria (Durck).

In sclerotic foci as well as in old scars, particularly in the peripheral submeningeal layers, there may be found large numbers of the so-called corpora amylacea (fig. 970), which normally appear in the brain substance after the thirtieth year of life (rarely earlier), and which are considerably increased in old age. They are rounded, egg-shaped or angular, concentrically layered or radiating bodies which stain like amyloid, both with iodine and the aniline dyes, (Siegert). According to Stroebe, Schmaus, Catola and Achucarro (lit.), and Sand, they come from destroyed axis cylinders or medullary sheaths; Siegert believes they are degenerated glia cells; Redlich, Nambu, that they are degenerated nuclei, while Sturmer (lit.) states they are not the products of glia, nor medullary sheaths, nor axis cylinders, but a general pathological metabolic product of the entire central nervous system, a catabolic product which, transported in the lymph stream, forms concrement-like structures. The bodies remain in the tissues for an indefinite time, and seem to be a sign of the using up of the nervous substance (Obersteiner); see lit. by Spielmeyer.

Calcification of capillary and arterial branches with thrombosis is occasionally seen in sclerotic foci. (This also occurs in gliomas.)

See Durck, and lit. by Thompson and Piney, concerning extensive calcification of one cerebral hemisphere.

Regeneration of the nervous parts of the brain does not occur in humans; at least, the weak indication of such processes (one isolated mitosis of a ganglion cell as seen by Saltykow, or the ingrowth of fine nerve

fibers into glial scars as shown experimentally by Borst) cannot replace lost substance (Pfeifer). Functional compensation is brought about only by vicarious activity of other similar centers and paths.

Circulatory Disturbances in the Brain

Anemia, Hyperemia, Edema.—The amount of blood in the brain varies throughout life. It is increased during intellectual work, and also in phlegmatic individuals. In hyperemia, the cerebral vessels are dilated, and the so-called perivascular lymph spaces become more or less empty from pressure; the lymph reaches the wide subarachnoid space, and may even be led into the peripheral lymph spaces as shown by Schwalbe in the lymph vessels of the neck. In anemia, on the other hand, the perivascular spaces are broader (Golgi). (Compare the views of Schaffer, p. 1806.) According to Held, there are intraadventitial lymph spaces, adventitial spaces, which, according to Spielmeyer, are the same as "Virchow-Robin's spaces," and which were erroneously believed to be between the media and adventitia—these occur in all the cerebral vessels. See also Evensen.

Anemia of the brain may be local or general. If the local anemia is complete, the result is ischemic encephalomalacia (p. 1894). In general cerebral anemia, the color of the white matter is flour white or blue white (in children); the section surface shows but few blood spots; the cortex is light gray, and less plainly separated from the medulla. Anemia may be acute or chronic.

Examples: acute anemia in death from hemorrhage; the brain is abnormally tough and dry. In chronic anemia, *e.g.*, chlorosis, pernicious anemia, leukemia, the brain is usually very pale. If the pressure within the skull is increased by intracranial tumors, hydrops of the ventricle and other things, the brain may contain but little blood. Acute brain anemia is caused, furthermore, by reduction in the blood flow in acute heart disease, or in narrowing of the cerebral vessels from vaso motor spasm. It occurs ex vacuo, when large masses of blood are sent to other parts of the body, *e.g.*, during the removal of ascitic fluid from the abdomen, in precipitant labor, by removal of large tumors; the symptoms are syncope and fainting. In Adams-Stoke's disease (p. 21), the cerebral symptoms such as loss of consciousness, spasms and apoplectiform convulsions are believed in some cases due to a temporary failure of the systole of the ventricle (heart block; Lichtheim, Schmoll and others; see also Kraus). Chronic anemia may be brought about by narrowing of the arteries in arteriosclerosis and syphilitic endarteritis.

Hyperemia.—Congestive hyperemia is found in the beginning of, and accompanying, acute inflammation of the brain. It is seen most

plainly when circumscribed, *e.g.*, in the neighborhood of an inflammatory focus. The cerebral substance appears specked or striped with red; the many punctate hemorrhages represent cross sections of filled vessels or small hemorrhages.

It should be kept in mind that arterial and venous hyperemia cannot always be differentiated in the cadaver. Even acute general congestive hyperemia cannot always be determined in the cadaver, though the symptoms during life may have indicated its presence. Occasionally, however, it can be recognized quite easily in the beginning of a meningitis, in sun stroke, delirium tremens, in the exaltation of the insane. The cortex is gray red; the white matter spotted red, and sprinkled with many punctate blood spots.

Passive Congestion.—In chronic passive congestion, the cortex is dark blue red or brown red, the white matter is rose red. Many dark red blood spots are visible on the surface, particularly in the white matter.

Stasis may occur in the general passive congestion of lung and heart diseases, or from local causes such as compression of the jugulars, the innominates, the superior vena cava, sinus thrombosis (fig. 904), tumors of the brain, particularly when there is pressure on the vena magna Galeni. The latter also produces an internal hydrocephalus. In marked active, as well as, passive hyperemia, the brain is voluminous; it fills the dura which becomes tense, and the convolutions are flattened. Occasionally the surface is dry and lustreless. Punctate hemorrhages are frequent. The weight of the brain is increased.

In edema of the cerebral substance, the brain becomes large, heavy, very pale and moist on section surface; it is saturated with serous fluid; the consistency may be so soft that there is pseudo fluctuation. Microscopically, the adventitial lymph sheaths are filled with fluid, and the meshes of the glia are stretched apart.

To recognize mild degrees, it is necessary to cut with a dry knife and observe if the blood flows away from the blood spots (sectioned veins) immediately, as it will when it mixes with the serous fluid flowing over them.

In more marked degrees, the medullary sheaths are swollen and varicose in the spongy tissue; the axis cylinders become convoluted and may be broken in parts. Destruction of medullary sheaths into myelin droplets and vacuolar swelling of the ganglion cells may also occur. The readiness with which degenerated nerves undergo swelling probably determines the swelling of the brain as a whole (Spiegel). The finding of granule cells prevents mistakes for post mortem softening or maceration (p. 1875).

Etiology.—Edema of the brain may be inflammatory, or congestive, and local or general. The edema of stasis occurs from the same causes as

venous obstruction. Local inflammatory edema accompanies various focal diseases of the brain such as hemorrhages, abscesses, tumors, infected gun shot wounds, etc., (Hart, lit.). General inflammatory edema occurs in the acute infectious diseases, disseminated meningeal tuberculosis, etc. Toxic edema, possibly due to toxic injury, and thus increased permeability of the vessel walls, appears, among other conditions, in chronic nephritis; according to Schnyder, in toadstool poisoning also. According to Kolisko, carbon monoxide poisoning and paralysis of the vessels also lead to edematous swelling.

The origin of the edema is often not determinable. In some cases it is probably an agonal symptom, inasmuch as a decrease in arterial pressure leads to venous stasis and this to increased transudation; sometimes it is quite probably of post mortem origin. The arteries are emptied and fluid is sucked from the subarachnoid space into the perivascular lymph spaces which are thus made wider. If the volume of the brain is diminished, as in marasmus, there appears an hydrocephalus ex vacuo, and the subarachnoid space, and finally the lymph sheaths become overfilled ex vacuo. The latter must also occur when the volume of the brain is diminished in general anemia.

True swelling of the brain (Reichardt, lit.) differs from edema and its swelling; the reason for the former is still obscure; as far as no new tissue (ameboid glia) can be demonstrated histologically, the thought is expressed that there is increased absorption of fluid in the cerebral substance itself. Cerebral swelling may, however, appear from the same causes which produce active increase in fluid, such as infection, intoxication, brain tumor; furthermore edema and cerebral swelling may be combined. (For more details and lit., see Reichardt; also under pseudo-tumor cerebri, p. 1938.) Panofsky and Staemmler, from their investigations, think it is impossible to reach the diagnosis of cerebral swelling through the "differential count" (see remarks, p. 1856), which was introduced by Reichardt; in spite of the advantage of Reichardt's method over simple weighing of the brain, they believe it possible to say only that cerebral swelling is probably, but not surely, present.

In passing, it may be well to mention the so-called colloid or gelatinous degeneration of the brain (Alzheimer, lit.), in which the vessel walls in the neighborhood are obstructed by hyalin masses in homogeneous or wavy forms; the latter stain intensively red with van Gieson's method, and some take the fibrin stain of Weigert; they may be sprinkled about so richly that parts of the brain take on a colloid appearance.

Cerebral Hemorrhage.—Most cerebral hemorrhages are spontaneous; they may be capillary or arterial.

Spontaneous venous hemorrhages of larger degree are very rare. In two cases, the author found that rupture of varices in the substance of the brain had led to fatal hemorrhage; the extent and amount resembled arterial hemorrhage; both cases occurred in eclampsia. See Beger for literature concerning the few cases of cerebral varix formation, in which 3, those of Amsler and Anders, involved the ophthalmic-meningeal vein. Other, equally large, cerebral hemorrhages in eclampsia are arterial (Wegelin), although arterial hemorrhages may also be punctiform in these cases.

Traumatic Hemorrhage.—A small minority of the hemorrhages into the substance of the cerebrum is of traumatic origin; their cause is quite varied, such as splintering of the bone in fractures of the skull, in gun shot wounds, stab wounds or after blunt traumata, concussion, falls. The latter usually produces concussion (red softening) with numerous punctiform, not widely scattered, hemorrhages, particularly in the cortex, but occasionally in the white matter also; the meninges covering the parts are infiltrated by blood, or are at least injured. If the vessels are atheromatous, traumatic hemorrhages may reach the size of a fist.

It is rare to find a traumatic tear of vessels in the central ganglia in which spontaneous hemorrhages are so frequent (Joerdens).

Even in the most severe concussion of the skull by blunt traumata, the hemorrhage and brain concussion may only be on the surface as in an observation of the author: automobile accident; after removal of the scalp, splinters of the skull, like pieces of a broken egg shell, could be lifted out, and the brain was removed without use of the saw (compare C. f. Schw. 1906, 10). The rare, larger traumatic hemorrhages with softening of parts of the brain substance may leave behind cysts reaching to, or into the ventricles (so-called traumatic porencephalia; see p. 1860). The author has seen a number of similar cases in children after compression of the skull.

The action of the so-called contrecoup, as after a fall on the back of the head, may be seen as yellow spots along the base of the frontal lobes. The small shattered places of the cortex are later changed to rusty or brown, yellow streaked, slightly depressed and slightly hardened defects, to which the thickened meninges are adherent. These "plaque jaunes," or scars, composed of a fine fibrous meshwork in which calcified ganglion cells and granular and crystalline blood pigments are present, are commonly found in old people with poor vessels and shrunken kidneys. They may, however, also be remnants of softening of the cortex following thrombosis or embolism. See p. 1894.

Spontaneous Cerebral Hemorrhages.—A necessary predisposing factor is a reduction in the resistance of the vessel walls; a vessel tears

because it is diseased. Frequently, the general blood pressure is high, but this alone can hardly tear a healthy vessel. There may be differentiated:

1. Punctiform hemorrhages coming from capillaries, veins or small arteries, often numerous, and as large as pin heads to peas, surrounded by larger, dark red blood spots. The very smallest hemorrhages may lie in the adventitial lymph spaces (false dissecting aneurysm), and merely compromise the surrounding tissue; somewhat larger punctiform hemorrhages, in contrast to blood spots, or droplets of blood issuing from cut veins, cannot be washed off, and infiltrate the substance of the brain. Not rarely, they form a ring around a vessel.

Punctiform hemorrhages occasionally occur in the neighborhood of fresh encephalitic foci (p. 1898), in embolism of arteries, in which fat embolism may be mentioned, in thrombosis of veins (see under sinus thrombosis), as an expression of a hemorrhagic diathesis in various infections and blood diseases, *e.g.*, pernicious anemia, leukemia, particularly acute, hemorrhagic purpura, etc. (see p. 1899). In the latter, the rounded or elongated, often sharply outlined, foci of blood may be punctiform, but they also reach the size of peas, occasionally walnuts, or larger, and the cerebral substance may be thickly sprinkled, whereas in infectious diseases such as anthrax, or malaria—in which latter condition they are thickest in the white matter of the cerebrum and the cortex of the cerebellum, (Durck; see Weingartner who also describes quinine hemorrhages)—typhoid fever, small pox, yellow fever, endocarditis, miliary tuberculosis, influenza (lit. by Kuczynski-Wolff), the hemorrhages are usually small, but may be very numerous, and usually accompanied by hemorrhages in the meninges. When very numerous, the condition is called purpura cerebri. In fat embolism, the blood spots are also thickest in the white substance, often very numerous in the corpus callosum, even though fat emboli are just as numerous in the cortex. According to Fromberg, the richer vascular supply of the cortex prevents the bleeding in these parts. Groendahl believes the circular hemorrhages are infarcts with anemic centers and hemorrhagic borders, but see Toeniessen. Microscopically, the condition is usually a diapedesis in the form of ring hemorrhages, (see under tuberculous meningitis, p. 1835) or rhexis with collections of

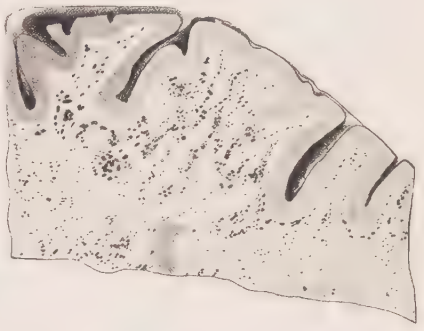


FIG. 931.—Innumerable punctate hemorrhages in the white matter in concussion of brain; man 41 years old; 7/8 nat. size.

blood (hematomas) in the adventitial vessel sheaths. Hemorrhages, even of considerable extent, may also occur through diaeresis, complete permeability of the vessels. In severe infectious diseases, fatty degeneration of the capillaries has been found. These, or damage to the capillary walls from bacterial emboli which are found frequently, have been held responsible for the hemorrhages. In the blood diseases, it is assumed that the permeability or brittleness of the capillaries has been increased, especially in leukemia, in which the vessels may be obstructed by the white blood cells (Marchand).

Purpuric spots in the white matter of the corpus callosum and the medulla following slowing of the blood stream, stasis, and diapedesis are believed by Ricker to be characteristic of poisoning by phosgene. Similar conditions have been found by Henneberg (lit.) in death from salvarsan; the chief rôle in this latter condition is played by damage to the capillary endothelial cells with the formation of hyalin thrombi, circumscribed necroses and diapedesis.

There are also cases in which multiple, sharply outlined, variably sized, punctiform hemorrhages are caused by a hyalin thickening and rolling up of the walls of smaller arteries and veins. Hemorrhages (hematomas) in the dilated lymph sheaths may be found, obviously caused by rolling up of the walls. Thus there arise complex pictures, particularly because many arteries in the centers of the hematomata contain thrombi, some organized and some recanalized. The author, for instance, saw this process in two cases, one in a 68 year old man with shrunken kidneys. His description of this case corresponds exactly to that reported by Weber.

2. Larger spontaneous hemorrhages, hemorrhagic apoplectic foci (figs. 932 and 933).

Apoplexy is the name given to the condition in which an individual falls unconscious. Since this is caused frequently by cerebral hemorrhage, the name apoplexy is often synonymously used with hemorrhage, not only in the brain, but in other organs, *e.g.*, the lungs. The term sanguineous apoplexy or cerebral hemorrhage is used in contrast to bloodless apoplexy caused by obstruction to vessels (p. 1895).

These occur from spontaneous rupture of a larger or smaller diseased blood vessel, often the seat of an aneurysm. The vessels most frequently involved are intracerebral arteries.

It is quite common to find changes in the cerebral arteries of persons past middle age; the larger ones are frequently sclerotic, atheromatous, and dilated; the smaller arteries are hyalin-sclerotic, and the capillaries hyalin, fatty degenerated or calcified. It is obvious, therefore, that arteriosclerosis in the intracerebral branches takes on a different form

from that in extracerebral branches. The aorta with its larger branches is not necessarily changed at all, and even though it is often diseased, it is not rare to find a striking inconsistency, *i.e.*, severe sclerosis of the aorta, and relatively good cerebral arteries, or vice versa. The source of the

FIG. 932.—Typical large spontaneous cerebral hemorrhage in l. lenticular nucleus with rupture into ventricles, which contain blood. (This horizontal section does not show the place of rupture.) Displacement of r. hemisphere; acute fatal apoplexy; man 52 years old; $\frac{4}{5}$ nat. size.

hemorrhage is often quite difficult to find; it proves to be either simple rupture of a vessel, or rupture of one or more aneurysms of middle size and small arteries, which, in fatal cerebral hemorrhage, are at least miliary in size, but often as big as lentils or peas. Charcot and Bouchard first pointed out that in spontaneous cerebral hemorrhage, these so-called

miliary aneurysms may be found in the hundreds, chiefly in the ascending branches of the middle cerebral artery which go to the optic thalami. They can be demonstrated after macerating parts of the brain in water or very dilute chromic acid solution, until the vessels with their aneurysms can be pulled loose from the tissues; according to Pick, this can best be done in a shaking apparatus. Charcot and Bouchard believed that these miliary aneurysms were true aneurysms and the source of sanguineous apoplexies; Ellis and Pick showed, however, that they are dissecting aneurysms, *i.e.*, blood sacs coming from damaged vessels following the hemorrhage (Dietrich; for more details, p. 142), and did no harm, *i.e.*, were not ruptured, but the pea-sized aneurysms are the true sources of the hemorrhage. The latter are therefore of direct importance in apoplexy, but they are false aneurysms or dissecting aneurysms as a result of simple rupture of a vessel, or are produced after the rupture of a previously existing dissecting aneurysm. Exceptionally, a single aneurysm as large as a pea is found in an otherwise unchanged vessel; this type is not, as a rule, due to arteriosclerosis, but to infectious processes. Old age, chronic nephritis, (see genuine shrunken kidneys, p. 1351), alcoholism, lues, gout, acute infectious diseases predispose to changes in the blood vessels. But, there may also be an hereditary tendency to apoplexy. In lues, and much more in shrunken kidneys, severe changes may appear in the vessels, even of young individuals. As favoring factors for the completion of rupture there may be mentioned, hypertrophy of the heart without dilatation, particularly hypertrophy of the left ventricle as developed in shrunken kidneys, and other chronic heart and arterial diseases which likewise raise the blood pressure. The strong heart breaks the weakened cerebral arteries (Wilx and Moxon). As immediate causes, inasmuch as they increase the blood pressure, may be mentioned: psychic irritation, too heavy a meal, bodily exertion, coitus (example, case of Gumprecht), strain during defecation, labor, coughing (whooping cough, see Neurath, lit.), convulsions during eclampsia, in which Orliansky believes that hypoplasia of the cerebral arteries is responsible for the fatal bleeding, etc. Rosenblath has evolved a theory all his own, especially to fit the hemorrhages of nephritics; he believes there is destruction of large parts of the cerebrum by the changed metabolism which brings about sudden fermentative and destructive activity; the hemorrhages then take place from the destroyed capillaries and veins.

If a large fatal hemorrhage with marked destruction of the brain is examined, extensive, loose, dark red coagulated and pulpy foci are seen in which particles of brain substance and blood clots are mixed; about the periphery there are tags of tissue and small torn vessels. The relations can be seen better after washing with water. The focus is completely

surrounded by a diffuse, blood stained zone, around which is a zone composed of punctiform to pea-sized hemorrhages. If the process is several days old, the neighborhood is swollen, edematous and stained by diffused blood pigment (lemon-colored edema); microscopically, false dissecting aneurysms can always be found, even at some distance from the apoplectic focus (fig. 8 on p. 133).

The sites of election of spontaneous cerebral hemorrhages are the larger central ganglia, particularly the corpus striatum and the thalamus,

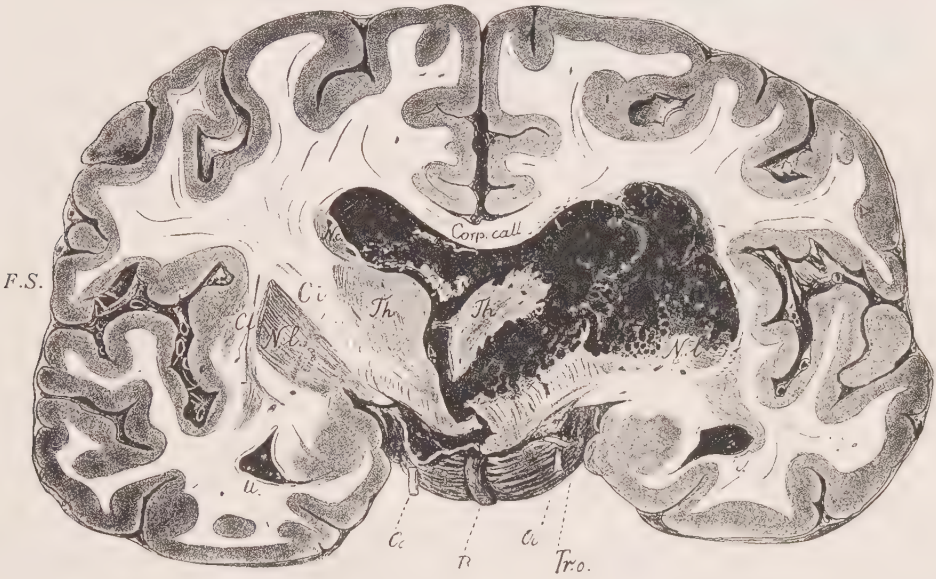


FIG. 933.—Cerebral hemorrhage with rupture into ventricles and into the subarachnoid space of the base; Corp. call. corpus callosum; Nc, caudate nucleus; Nl, lenticular nucleus; Ci, internal capsule; Th, optic thalamus destroyed by the hemorrhage. Remains of fornix below the corp. call; the soft commissure visible in III ventricle; inferior cornua full of blood; Oc oculomotor; Tr.o, optic tract; B, basilar artery; F.S, fossa of Sylvius; Cl, claustrum; man 71 years old; $^{11}/_{12}$ nat. size.

and the neighboring medullary strands of the internal and external capsule, which are supplied by branches of the artery of the Sylvian fossa (end arteries). The blood pressure is higher here than in the cortex, into which sink branches of the meningeal arteries, which are relatively smaller and possess anastomoses. Next to the basal ganglia and their vicinity, the point of election is the centrum semiovale. More rarely the pons, medulla oblongata and the cortex are chosen. The hemorrhage often ruptures from the central ganglia into the ventricles, and the blood streams out into the subarachnoid spaces of the base of the brain. Rarely, rupture takes place through the cortex towards the surface, the convexity or the base (fig. 933); the subarachnoid spaces become filled with blood, and the surface of the brain is covered as by a cap. In the pons, there are

occasional hemorrhages from branches of the basilar artery which dissect their way along the nerve bundles and are hindered from spreading by the resistance of the neighboring bundles; these dissecting hemorrhages may spread into the crus cerebri. But when death has occurred in a few hours, the pons may be found swollen and filled by a mass of blood so that it fluctuates to the touch. The thin layer of nervous substance surrounding the blood may also be torn; then the hemorrhage reaches the fourth ventricle, or the meninges, *i.e.*, the subarachnoid spaces.

Stripes of hemorrhage also occur in the pons in traumatic brain rupture with intact skull (Kolisko), and are caused by forcing downward the contents of the posterior fossa, which leads to tearing of the blood vessels which enter the crus cerebri and the pons from the base. They also occur following traumata to, and spontaneous hemorrhages into, the cerebrum, when a sudden, very marked increase occurs in the intracranial pressure.

The extent of the hemorrhage is variable; in extreme cases an entire hemisphere may be saturated; it usually occurs in only one hemisphere and rarely in several situations at one time. On the other hand, it is not rare to find a number of hemorrhagic foci of different ages. In addition to an extensive hemorrhage, there are occasionally innumerable, white, encephalomalacic foci, or colorless, or pigmented cysts, sclerotic around their edges.

Consequences of hemorrhagic (apoplectic) foci. Hemorrhages which break from the central ganglia into the lateral ventricles, those in the pons and in the medulla, are often fatal in a very short time (fulminating fatal course), or death occurs in coma. In other cases, the attack passes over, to be repeated a number of times. If we except those hemorrhages which lead to death through destruction of vital centers such as the respiratory and cardiac in the medulla oblongata, another cause of rapid death in profuse cerebral hemorrhage is acute increase in intracranial pressure producing anemia of the brain from diminished blood flow (adiamorrhysis, Geigel); this is manifested at autopsy by marked flattening of the bloodless convolutions. It also explains the apoplectic insult. The increased intracranial pressure may abolish the functions of the vital centers, and a hemorrhage into the basal ganglia, which of itself would merely lead to paralysis of the arm, tongue, etc., may end fatally because of the increased brain pressure. If no signs of brain pressure are obvious at autopsy, and if the site of the lesion does not explain the acute death, the tendency is to ascribe the condition to shock, or concussion of the brain with reflex paralysis of the centers; an individual wounded by a pistol bullet which has not passed through vital parts, or by a blow behind the ear, can die from shock. The increased cerebral pressure is helpful

to a certain extent, for when the external pressure reaches the pressure in the ruptured vessel, the bleeding stops. Coagulation also obstructs the source of bleeding.

Of the focal symptoms of hemorrhage we will mention the most frequent. Since the sites of election of hemorrhages are in the central ganglia and their neighborhood, a hemiplegia or unilateral motor paralysis develops in most cases, because the pyramidal tracts which run through the internal capsules are directly or indirectly destroyed. The anterior or middle parts of the posterior division of the internal capsule, between the optic thalamus and the lenticular nucleus, contain the motor paths of the opposite half of the body. Destruction of these produces cerebral hemiplegia, *i.e.*, paralysis of the legs, arms, musculature of the face, particularly of the lower territory supplied by the facialis; the mouth is pulled toward the healthy side, and the protruded tongue (hypoglossus) falls away from the paralyzed side. In the posterior third of the posterior cornu of the internal capsule is the course of the sensory paths; if these also are involved, the result is hemianesthesia. If the hemorrhage has not directly destroyed the motor paths, but merely damaged them indirectly through compression, the paralysis may disappear (partially) when the hemorrhage is finally absorbed. Then the hemiplegia takes on the character of an indirect focal symptom. But even when the hemiplegia produces direct focal symptoms, some ability to move returns, particularly in the legs, while the arm usually remains paralyzed and almost unusable. See under spinal cord and muscles, concerning descending degeneration of the motor paths and the consequent contraction of the paralyzed muscles.

The Course of Hemorrhagic (Apoplectic) Foci.—Smaller foci are composed of coagulated blood which encloses the destroyed brain substance and appears dry, since the serum is soon pressed out. Even larger foci which at first exert marked pressure on the neighborhood soon begin to dry and shrink from coagulation and squeezing out of the serum which is removed through the lymph stream. Many red blood cells are removed by leucocytes, others are hemolyzed; the hemoglobin is taken up by white blood cells and others, or is transformed into brown, wavy or granular hemosiderin pigment in loco, or into granular or crystalline iron-free hematoidin. Free pigment is found earliest on about the eighteenth day and always from the sixtieth day onward (Durck). The particles of brain are softened and become granular; these are removed by cells and transported further (fat granule cells, see p. 1875). The interadventitial lymph spaces of neighboring vessels are filled with granules and granule cells. This changes the consistency of the focus; it becomes pulpy and fluid, homogeneous; in 20 days its color is brown red or sienna brown to

chocolate colored, then rust brown to leather yellow. In a few cases, a marked reactive inflammatory softening is established in the neighborhood; glia is produced from the neighborhood, and a small focus is finally completely filled in, but a larger mass is merely encapsulated by connective tissue. After the products of destruction are removed (months), there remains a cystic space filled with serum (apoplectic cysts), usually lined with a soft brown myxomatous connective tissue surrounded by thick, tough, vascular fibrous connective tissue; in other cases the chief mass of the cyst wall is glia and the formation of connective tissue regresses. The cysts can be called "degenerative."

For histological details, see v. Monakow, Claude and Loyez, Spielmeyer.

On section, these cysts may be single, or multilocular because of the blood vessels contained inside, which have escaped softening and about which newly formed fibrous strands are criss-crossed, giving it the appearance of a sponge. The inner surface is usually dark red or brown from blood pigment; it takes many years for the pigment to disappear completely, whereupon the cyst is quite similar to one arising from white softening. The contents are usually serous, water-clear, more rarely, cloudy brown or milky, and mixed with fat granule cells. The size varies from a pea to a walnut; the majority are small; a few reach considerable size. In a case observed by the author in a boy, who, a number of years before, had received a blow on the head from the flails of a windmill, there was a brown pigmented cyst as large as a hen's egg in the white matter of one hemisphere. Occasionally, the cysts remain permanently, especially if large; the walls of smaller cysts may adhere and obliterate the cavity.

The defects of cerebral hemorrhage may also be cicatricized; the scar is chiefly a product of growth of the glia; it is pigmented brown and may contain a nucleus of thickened, brown yellow, cholesterin-containing material. If the walls of an apoplectic cyst become adherent, there finally appears an elongated, sometimes radiating, hard scar.

Aneurysms in the larger extracerebral, intracranial arteries are frequent along the base. They usually occur in youthful or middle aged individuals, are often accompanied by endocarditis or lues, and relatively rarely, by simple arteriosclerosis. There are other cases in which nothing of etiological moment can be found except an obscure perivascular inflammation. According to Ponfick, these are often of embolic origin. Reinhardt believes there is a defective congenital anlage in some cases. Direct traumata such as fractures of the skull, stab wounds, gun shot wounds (see Lutz), and indirect traumata including injuries to the head, or to the cervical vertebral column, even if inflicted years before, may lead to aneurysms of the vertebral and the basilar arteries; the author has seen

such cases in which there was no trace of general arteriosclerosis (Saathoff, Hedinger, lit.). The aneurysms are mostly saccular, small (fig. 5, p. 133), usually as large as peas, seldom as big as cherries, but have been reported as large as hen's eggs; they are most often single, but may be multiple. Diffuse arteriosclerotic aneurysms are quite common in the basilar and vertebral arteries, but are without much importance. They are most frequent in the basilar arteries, then in the middle cerebral artery, and in the anterior and posterior cerebrals. Busse calls attention to the particular anatomic relationships causing aneurysms of the anterior communicating artery (comp. p. 137). Rupture of a latent aneurysm following marked increase in blood pressure usually gives rise to fatal hemorrhages; an aneurysm may, however, become plugged and later undergo re-perforation (Wichern); the subarachnoid space, in which the basilar cerebral arteries are found, is tensely filled with blood. The base of the brain is covered by a thick coagulated pillow of blood, in which the ruptured aneurysm may be hidden. If this tensely stretched pillow is broken, the free hemorrhage enters between the arachnoidea and the pia; hemorrhage into the brain substance may also occur, particularly from an aneurysm of the Sylvian fossa artery. Spurius aneurysm may complicate true aneurysm; sudden rupture of the former produces the picture of cerebral apoplexy and sudden death (see E. v. Hofmann, Wichern, lit., Reinhardt). In such events, the subarachnoid space may be tensely stretched; in primary intracerebral hemorrhages, however, this is not the rule. See Lebert; lit. by Roth, Kurschinsky, concerning the symptomatology of aneurysms of the cerebral arteries, which are differentiated with difficulty from brain tumors.

Embolism, Sclerosis and Thrombosis of Cerebral Vessels and Their Consequences.—Softening of the brain (encephalomalacia) may be brought about by various causes. It is an expression of acute inflammation of the cerebral substance (encephalitis) as well as of disintegration of the parts following traumata or nutritional disturbances caused by thrombotic and embolic obstruction of vessels. See pages 1870 and following, concerning the histological processes in the nerve fibers and ganglion cells and in the vascular interstitial tissue which follow death of cerebral substance.

Sudden or granular obstruction of a cerebral artery may occur from embolism or autochthonous thrombosis.

The arteries of the basal region, which supply the base of the brain, are end arteries (Cohnheim). Those of the cortex of the brain anastomose as long as they arborize in the pia; after turning at right angles they enter the cortex, as fine branches. The cortex is more readily supplied by collaterals than are the central ganglia and the centrum semiovale; thus,

e.g., obstruction of one artery of the Sylvian fossa produces no disturbances in the cortex, while there are always disturbances in the white matter. Five chief branches come from this artery: (1) to the third inferior frontal convolution, particularly its posterior part; (2) to the anterior; (3) to the posterior central convolution; (4) to the lower parietal lobe, and the upper part of the first temporal convolution; (5) to the lower part of the first and second temporal convolutions—and in addition, it supplies three deeper branches to the basal ganglia and to the largest part of the caudate nucleus, the lentiform nucleus, the internal capsule and a part of the optic thalamus. On the other hand, the entire area supplied by this artery may be softened, when the first 2 cms. are occluded.

Emboli come mostly from the left heart, (endocarditis, among other conditions, lit. by Savaliew); when the foramen ovale is open, they may also come from the right side (crossed embolism). Diseases, (endocarditis), of the mitral and the aortic valves, parietal thromboses in the auricles and auricular appendages, as well as aneurysms of the heart are (in general) the most frequent source of emboli. Other points of origin of brain emboli are: atheromatosis, aneurysms, and even mild fatty degeneration of the intima of the ascending aorta, (see case, p. 102), and the branches to the cerebrum, particularly the bifurcations of the carotids, more rarely from the veins of the lungs in cavity formation, gangrene, etc. See p. 1893 concerning embolism following ligation of the carotid, or in aneurysms of this artery. An autochthonous thrombosis of an atheromatous or an aneurysmal intracranial cerebral artery or a traumatic thrombosis of the internal carotid before it enters the skull, may also lead to embolism in the smaller branches. The entrance of an embolus is occasionally directly due to an acute increase in blood pressure during labor, coitus, etc. The emboli always remain fixed at a bifurcation; they are pale gray red to colorless, fairly firm, occasionally partly calcified, and if present for a short time, are more or less adherent to the vessel walls. Secondary soft red thrombi often spread into the lumena of neighboring ramifications. Embolism is more frequent on the left than on the right side, which is explained by Wegelin on the basis of the left common carotid artery coming direct from the arch of the aorta and therefore being more directly in the path of the aortic current; the right carotid leaves the innominate artery which makes almost a right angle with the arch of the aorta. The female sex is more frequently the victim.

For air embolism, see p. 207. Fat embolism, p. 386.

Thrombosis of cerebral vessels occurs in disease of the vessel walls which is very frequent (p. 1884). The types are ordinary senile sclerosis and atheromatosis, or this condition brought about by shrunken kidneys, lead intoxication, alcoholism, etc., as well as syphilis which leads to

narrowing and obstructing endarteritis. Gummata (more rarely tumors, or larger tubercles, or tuberculous meningitis –see p. 1836), are particularly common around vessels, and lead to thrombotic occlusion. Injuries or ligation of the common carotids, or, more frequently, the internal carotid, lead to no symptoms at all, temporary symptoms, or to softening of the brain and hemiplegia (Ranzi, Rohardt and others); however, a progressive thrombosis may follow, or loose secondary emboli are distributed in the branches, or the artery of the Sylvian fossa. The same train of events has

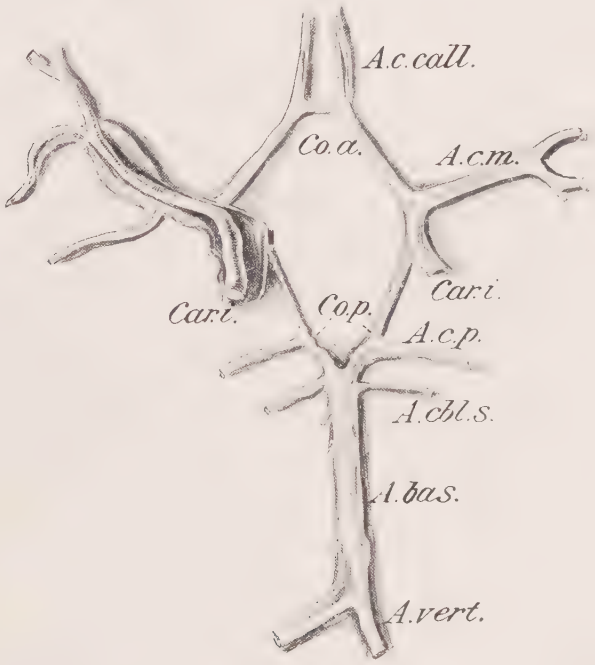


FIG. 934.—Embolism of l. intern. carotid artery (car.i) and artery of fossa of Sylvius (a.c.m.). Woman 49 years old with thrombus in l. auricle of heart; softening of l. cerebral hemisphere; A,c. call. Artery corp. callosi (anterior cerebr. artery); A.c.m. median cereb. art (A fossa of Sylvius); A.c.p, posterior cerebr. artery (deep cerebr. art.); A bas, basilar art; A. vert, vertebr. art; Co.a, anterior communic. art; Co.p, post. commun. art.; nat. size.

been seen by the author in traumatic aneurysms of the carotids, (Lexer, Stierlin and v. Meyenburg). Thrombosis may also result from various acute infectious diseases. Embolism readily occurs from thrombosis of the cerebral vessels provided the thrombus is not tightly adherent and completely obstructing. In smaller vessels the differentiation between embolism and thrombosis is quite difficult or impossible.

If a collateral circulation does not come to the rescue after obstruction of a cerebral artery, the result is ischemic necrosis and, soon after, softening of the parts supplied by the artery (encephalomalacia). Occasionally, blood enters the dead area from neighboring vessels; this leads to a sort

of false hemorrhagic infarction; the dying tissue is infiltrated by blood and thus discolored (red softening); when the blood coloring matter is changed, the softening is brown or yellow. If no blood leaves the neighboring vessels—which is usually the case,—the condition is spoken of as white (ischemic) softening.

The rapidity with which the blood flow is obstructed determines to some extent the fate of the area of brain involved. In sudden, embolic, total obstruction, all changes naturally appear rapidly. In gradual obstruction, as, *e.g.*, embolism followed by secondary, totally obstructing thrombosis, or primary thrombotic obstruction, the regressive changes occur more slowly and in proportion to the gradual obstruction.

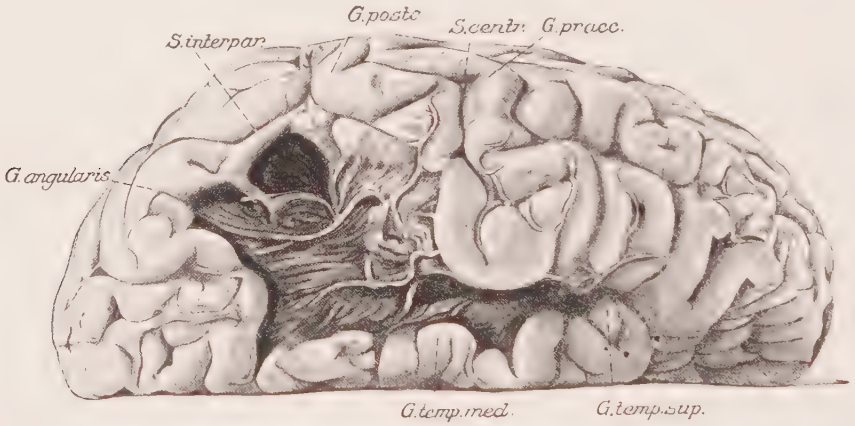


FIG. 935.—Large old anemic area of softening in r. cerebral hemisphere. Deep depression lined by folded adherent soft meninges, corresponding to lower parietal brain and greater part of upper temporal convolution of man 77 years old. Arteriosclerosis of cerebral arteries; somewhat less than $\frac{2}{3}$ nat. size.

Soon after sudden obstruction of a vessel, the part of the brain undergoing anemic softening becomes fleshy, spongy, and, following serous saturation, of white color and more voluminous; it is not sharply outlined from the neighborhood. Occasionally, fresh foci are pulpy and yellow from the imbibition of dissolved blood coloring matter. Microscopic signs of softening are found after two days (myelin and fat droplets, fat droplet cells); ordinarily the focus becomes white to white blue, soft, brittle; it may be peculiarly dry, and finally granular, similar to a cut of dry cream cheese; on section, masses adhere to the knife. Later (in weeks, occasionally earlier) the situation becomes semi-fluid, like milk of magnesia, and is composed of products of cell destruction and fat granule cells suspended in a watery fluid. There may appear true disintegration cysts, the contents of which flow out on section and leave a ragged hole. In foci which are themselves totally liquefied (simple necrosis), the blood

vessels are also entirely destroyed; in other cavities they are retained as a network, partly collapsed, partly filled with detritus, and partly open. The fluid gradually becomes more viscid through resorption. The contents become clear like that of a cyst, or remain cloudy following progressive destruction of tissue. In older foci the walls become hardened (see p. 1877), and the vessels stretched across the focus become thickened. Granule cells can be found for long times in the periphery of foci; they are evidences both of earlier tissue destruction and of reparatory processes. While white encephalomalacic foci in the medullary substance frequently become cysts, it is a much less common event in the cortex; in the latter, there are chiefly peripheral red softened foci, which, with the exception of those in fetal and children's brains, are less prone to undergo total liquefaction than the anemic foci. Rather they become rust brown, later yellow, fat granule cell-containing areas which finally sink below the surface as they harden through the overgrowth of glia into rust brown dense or cavernous scars. The underlying white matter is often tough and yellow under the sunken, brown cortical scar ("plaque jaunes," Charcot).

The extent of encephalomalacic foci varies; they may be very large, *e.g.*, when the internal carotid is obstructed by a thrombus which extends from the bifurcation of the artery to the corpus callosum and the artery to the Sylvian fissure. If the cortex is softened, the swollen convolutions are flattened against the cranium and against each other. Frequently, the foci are small but numerous; if they become old, the brain appears moth-eaten, or sieve-like, or perforated like cheese. Older cortical foci are sunken, but may still allow recognition of the convolutions; they are tough, but elastic; the meninges covering the place are hardened, adherent, white yellow or brown, and richly vascular.

When arteriosclerosis of the cerebral arteries is the basis of mental diseases, among them, arteriosclerotic dementia, there are a number of foci of cortical damage caused by the sclerosis. They take the form of: (*a*) completely softened foci, in which the entire functioning and glious tissue over larger or smaller areas is completely destroyed, and in which scars and cysts are end results. The masses are often wedge-shaped with the base at the surface of the cortex. (*b*) Incomplete softening (Alzheimer) with retention of the glious interstitial tissue, is another finding, while the more sensitive nervous elements undergo disintegration; many granule cells appear. Glious scars and thickenings, as well as cysts may be the end results of complete as well as incomplete softenings ("porose cerebrale," P. Marie). (*c*) Light spots, the so-called "light" areas of the cortex, may not be visible macroscopically; they contain a central sclerotic vessel, and capillaries with hyperplastic or degenerated endothelial

linings. In these foci, which are merely a particular variety of (*b*), (Schroder), there appear seriously damaged, but, structurally intact, nervous cells, with gross disappearance of the finer nervous structures; the glia shows little or no change; granule cells are lacking. The process is therefore merely a simple rarefaction of the functioning nervous tissue without secondary growth of the supporting framework, caused by disturbance of the vascular supply (Schroder). This condition, described first by Borst (see also Arndt), was believed by him to be due to primary stasis of the lymph stream, which destroyed the nervous elements but spared the glia; the latter filled out the spaces formed by the destruction of the former. Alzheimer and Saltykow, on the other hand, believe that sclerosis of vessels is the primary condition (Schmaus).

There also occurs a late epilepsy due to general arteriosclerosis of cerebral vessels (for the types of epilepsy, see Alzheimer). Also see A. Jacob concerning arteriosclerosis of the nervous system.

These particular arteriosclerotic changes in the brain are not to be confused with those due to old age. As Binswanger and Schaxel emphasize, in presenile dementia as well as in a number of other mental diseases, the hypoplasia of the cerebral arteries as described by them is due to a constitutional factor; and in presenile dementia, arteriosclerotic changes may not take place even if the sufferer lives to old age.

In addition to obstruction of arteries as a cause, red softening may be brought about by inflammations (inflammatory red softening, encephalitis), and by thrombosis of the veins (see sinus thrombosis, p. 1807). The parts are more voluminous, softer, diffusely red and sprinkled with punctiform hemorrhages; later they soften to a pulp (see fig. 904, p. 1807).

Microscopically, the non-inflammatory red softenings show products of the disintegration of nerve fibers, ganglion cells, glia and vessels, all of which undergo early fatty degeneration. Capillaries are filled with fat droplets and pigments. The red blood corpuscles undergo the changes described on page 1889. If the focus becomes yellow, the condition is known as yellow softening (p. 1894). Later the foci become cysts or pigmented scars. In inflammatory red softening, there is added an infiltration of the vessel walls and the emigration of leucocytes (see encephalitis, p. 1897).

The consequences of obstruction to vessels are many and varied, but will be only briefly discussed. In sudden embolic occlusion of a larger vessel, there is, as a rule, a sudden apoplectic insult. The coma is usually of milder intensity and duration than in sanguinous apoplexy. Among the paralyses, there is typically a hemiplegia (obstruction of the trunk of the artery to the Sylvian fossa), which, if of the right side of the body, *i.e.*, when the left Sylvian fossa artery is obstructed, is accompanied by

total or partial aphasia (the third left temporal convolution, Broca) as a direct focal symptom. While doing autopsies, it is well to remember that hemiplegias or hemipareses occur in which no macroscopic changes can be found. This is true of uremia, alcoholism, diabetes, lead poisoning, arthritis and pneumonia. Oppenheim and others saw it in the cachexia of carcinoma; Oppenheim expresses the opinion that it is a toxic focal disease; see also Finkelnburg. (This fact is also important in the diagnosis of brain tumors in which surprises are not uncommon.) Monoplegia is also quite frequent, since the focus may be confined to a circumscribed area of the cortex. Obstruction of the artery cerebri profunda is usually followed by hemianesthesia and hemianopsia. If obstruction of the internal carotid is not compensated by collaterals, or if the thrombosis extends to the anterior and middle cerebral arteries, there results hemiplegia with deep coma and quick death. Thrombosis of the basilar (and vertebral) arteries and their branches, produces acute (apoplectic) bulbar paralysis, which leads to death in a few days or weeks from aspiration pneumonia, paralysis of the respiratory and the cardiac centers. We cannot discuss in further detail the symptoms caused by softening of other focal parts. In general, however, it may be remarked that softening sometimes occurs in situations in which typical spontaneous hemorrhages do not, as a rule, appear, as *e.g.*, in the cerebral cortex, and in the medulla of the cerebral hemispheres.

Inflammations of the Cerebral Substance, Encephalitis

Acute, Non-suppurative Encephalitis.—This occurs (*a*) following trauma; (*b*) secondarily by the hematogenous route in various infectious diseases which are frequently accompanied by meningitis, thus in pyemia, endocarditis, cerebrospinal meningitis, spotted fever (p. 122), anthrax, rabies, sleeping sickness (p. 1869), influenza, typhoid fever, articular rheumatism, measles, scarlet fever; (*c*) by continuity from an inflammation in the neighborhood, as from the middle ear, the nose—thus, in glanders the inflammation often takes on a hemorrhagic character and involves the soft meninges as well,—or by continuity from an already existing disease of the brain itself such as softening, hemorrhagic foci, tumors, parasites, etc. The interstitial vascular connective tissue apparatus is the point of origin of the inflammation, and there follows a degeneration of the nervous structures. The result of an inflammation is softening, which may be red or yellow or white. The most frequent organisms are pyogenic cocci and the diplococcus pneumoniae. Consult Homen for infectious-toxic, non-suppurative encephalitis.

In carbon monoxide, or illuminating gas poisoning, late, or even early changes occur in the central nervous system, which, among others, are

presented as a disseminating "encephalitis," and which, in protracted carbon monoxide poisoning (Kolisko), are accompanied by ischemic degeneration, sharply outlined encephalomalacic foci, and also hemorrhages (Chiari).

The suspicion arises of a primary toxic effect of the carbon monoxide (lit. by Hedren and Sibelius); compare also p. 1912. There is a striking specific affinity of carbon monoxide for the central ganglia, especially the nervous tissue of the globus pallidus, *i.e.*, the middle portion of the lenticular nucleus; symmetrical foci appear in these regions as well as in the vessels (Stewart, Kolisko, Ruge). Wohlwill states that, among other severe regressive changes in the arteries, particularly in their mediae, he has found calcification even in three days, (see Herzog), and he emphasizes particularly the relation of softened masses to changes in the blood vessels.

Anatomically, the inflammatory complex is composed of regressive, as well as of reactive exudative, hematogenous and lymphogenous proliferative changes.

The encephalitic foci are always circumscribed and often microscopic in size, most numerous in the cortex; they show infiltration of the vessel walls, with foci of lymphocytes and more or less numerous leucocytes in the perivascular spaces and their neighborhoods, and in the tissues. Signs of disintegration of the nervous structures are present, and granule cells are found.

Occasionally, extensive foci of white or yellow or red color are found, in the neighborhood of which the cerebral substance, which fluctuates to the touch, is swollen, moist, soft and pulpy; an entire hemisphere may be involved. Larger masses usually show punctiform hemorrhages in their peripheries.

If healing takes place, restitutio ad integrum may follow, but rarely, and only in the first stages; usually the nervous structures in the vicinity of the foci are entirely or partially destroyed and the parts are sclerosed. This end result of an acute encephalitis in the motor parts of the cortex is probably at the basis of some of the cases of cerebral paralysis of children (p. 1869).

According to Eug. Fraenkel, bacterial masses can be found without macroscopic changes in the meninges and the brain of almost 50% of the cases of acute infectious diseases; this is particularly true of the pyogenic organisms, but not for diphtheria bacilli and only in mild degree for typhoid organisms. They may produce thrombosis in cerebral vessels, collections of cells in the perivascular lymph spaces, pigment masses in the latter, exudative and proliferative changes in the meninges, and small extravasations and necroses in scattered situations. All these lesions are, however, mild, and usually heal with complete restitution of the parts.

Cases of acute encephalitis are also observed which showed various cerebral symptoms during life, but which, at autopsy, show no macroscopic changes (see O. Meyer). According to Moser (comprehensive lit.), chorea minor is due to infectious-toxic causes (lit. by Sand); and staphylococci, which produce small inflammatory foci as well as areas of softening in the cortex, are important, because they possess direct neurotoxic affinities.

Occasionally the inflammation takes on a hemorrhagic character (acute hemorrhagic encephalitis), and the cerebrum is richly sprinkled with tiny hemorrhages (O. Meyer, lit.); in other cases, particularly those following influenza, the hemorrhages are larger, symmetrical, and combined with foci of softening, usually in the cerebrum; occasionally, they rupture into the ventricles (M. B. Schmidt). Even apoplectiform hemorrhages have occurred into the ganglia of the cerebrum (Nauwerck). Sinus thrombosis may be an accompanying condition. Since this hemorrhagic encephalitis (Friedmann) may be an independent, acute, rapid, but not necessarily fatal disease (Strumpell), there is possibly a relationship to cerebrospinal meningitis (abortive encephalitic form, or meningitis?; Oppenheim, lit.). More frequently, however, it accompanies such acute infectious diseases as influenza (Leichtenstern, Furbringer, Nauwerck, Mittasch, comprehensive lit.); whooping cough (lit. by Hada); appendicitis (Heusser); diphtheria, typhoid fever, pneumonia, cerebrospinal meningitis (lit. by Laignel-Lavastine, Voisin); and pure infections with malaria, in which the hemorrhages are in the medullary substance (Durck, Seifarth). In the type described by Wernicke (acute hemorrhagic superior polioencephalitis, probably related to alcoholism), inflammatory hemorrhagic foci are localized to the central gray matter of the third ventricle, the aqueduct of Sylvius and occasionally to the fourth ventricle. M. B. Schmidt (lit.) differentiates pure purpura of the brain, and hemorrhagic encephalitis; the former is the only way in which the inflammation is expressed; the latter is accompanied by inflammatory exudative processes. (See also lit. by Oppenheim-Cassirer, Oeller and Langbein, Oeller.)

See Socin, Herzog, Fahr, among others, for the encephalitis or hemorrhagic myelitis and death following administration of salvarsan.

Since the communication of v. Economo (1917), great interest has been expressed in non-suppurative **encephalitis lethargica,** which is recognized as a disease sui generis, and is also called polioencephalomyelitis and epidemic encephalitis. The macroscopic changes in the brain are relatively minor; increase in its weight, hyperemia, and occasionally saturation of the meninges, richly vascular, moist, section surfaces, and occasional grayish red staining of the gray substance are found. Histologically, the early stages show marked filling of the meningeal ves-

sels and perivascular infiltrations of lymphocytes, which spread to the cerebral substance; Hauptli, by the oxidase reaction, found leucocytes also in this tissue infiltration in the first days. Plasma cells arising from adventitial cells (Herzog) appear in later stages, while the lymphocytes become few in number; in both white and gray substance are foci of perivascular glious overgrowth with destruction of ganglion cells, and neurono- or neurocytophagia. In later stages, the foci of necrosis are extensive and contain many granule cells with focal overgrowths of glia, (cells and fibers); according to Hauptli, these are reparatory processes connected with the removal of the damaged parenchyma. The foci lie chiefly in the brain axis (pons, see Reinhardt), particularly in the neighborhood of the third and fourth ventricles (Dietrich, Siegmund). Foci, however, are present also in the spinal cord, and according to v. Economo, particularly in the anterior horn, in greater number in some epidemics than in others. (For more details, see Gerlach, lit.)

Etiology.—Encephalitis "lethargica," (there also occur non-lethargic forms), is believed by most authors to be a disease sui generis, which must be separated from la grippe, even though la grippe may be present before, or at the same time the encephalitis manifests itself; there may be some sort of relationship between these diseases (see, *e.g.*, Tobler). The causative organism is believed by many, thus v. Economo, to be the diplostreptococcus pleomorphus (von Wiesner); Dietrich found it five times in 20 cases; but the specificity of this organism from the pneumostreptococcus (pneumococcus) is doubted, and it is found in various organs in grippe, as well as in grippe encephalitis (Bernhardt). Others believe a filtrable virus is responsible (Mowes, lit.), similar to that which is considered the cause of anterior poliomyelitis (see under spinal cord), a disease which shows marked histological similarity to encephalitis lethargica (Hauptli, lit.). Dietrich believes both processes are fundamentally similar, the difference being in the parts involved. (The very extensive literature concerning this question which is still awaiting solution cannot be discussed further; a few more important, particularly pathological-anatomical, references will be found in the literature in the appendix.)

Rabies, in addition to general hyperemia, produces hemorrhagic or white soft inflammatory foci in the brain and spinal cord somewhat similar to those of poliomyelitis. Leucocytes may be collected into miliary abscesses in the neighborhood of vessels. The changes are most severe in the medulla oblongata. The virus is found in the central nervous system and in the salivary glands from which it is said to be transported via the chorda tympani. The toxins spread from the point of entrance along the nerves to the brain and spinal cord. Concerning the specific Negri bodies (neurocytes hydrophobiae), which are found first in the

ganglion cells of the hippocampus major, and finally, in the ganglion cells of the medulla of the adrenals, (see lit. by A. W. Williams and M. M. Lowden and particularly Frosch), the latter, with Babes, believes that they are not the causes of the disease; Frosch, furthermore, believes they are not parasites, but thinks that their demonstration is the best, quickest, and most certain method of diagnosing rabies (see also Volpius, Sanfelice, lit.). Babes, on the other hand, believes they are encapsulated forms of parasites. Lentz calls them products of the ganglion cell reaction to degenerative processes caused by the influence of the organisms on these cells; Piron holds that Lentz's "passage bodies" are accidental products of cell destruction which may be found in various other infections. Sanfelice is of the opinion that Negri's bodies, which he found in some animals during their winter hibernation (hedge-hogs and others), are nothing other than changed granule bodies which have entered the cytoplasm of cells; he considers the virus is, in reality, a toxic cell product. But Negri (lit.), confirmed by Golgi and Grassi, demonstrated a sort of life cycle in the parasitic, in fact, protozoal, nature of these bodies. Koch and Rissling, on the other hand, are inclined to Babe's opinion. See Costantini and Buschi concerning the changes in the nervous structures caused by tetanus toxin. Simple method for the examination of fresh tissues: section through the hippocampus major, light scraping of the knife releases a detritus containing many ganglion cells; examination of this material after addition of a drop of very dilute acetic acid allows recognition of the parasites (Negri-Luzzani). For staining methods, see Stutzer; (Loffler's methylene blue, differentiation with 1% tannic acid, paraffin embedding). See lit. in the appendix concerning the resemblances of rabies and poliomyelitis.

Encephalitis neonatorum is a term used by Virchow to describe a disease found particularly often in syphilis, in which fat granule cells appear in the cerebral substance profusely or in masses, producing yellow white, cloudy spots; these contrast sharply with the transparent, gray red, medullary substance which at that time has scarcely obtained its medullary sheaths. According to Jastrowitz, however, granule cells in the brain are physiological from the fifth month of pregnancy to the eighth extrauterine month, and are related to the formation of the medullary sheaths of the nerves. It is, however, questionable if all such foci can be explained in this manner, since they are soft, and occasionally reach the size of lentils to peas, and, furthermore, may contain round cells and hemorrhages. Wohlwill differentiates between fat granule cells of pathologic catabolic import which represent the reaction of the nervous system to various damages, and anabolic cells which are related to the progressive formation of medullary sheaths and occur typically up to the

sixth month of life. In addition to those in syphilitics, similar foci are found in newborns whose head has remained long in the birth canal, and in severe icterus accompanied by suppurations of the umbilical cord (Birsch-Hirschfeld), or in children whose mother had eclampsia and who themselves had convulsions. Fischl, Cellen, who emphasize the focal fatty degeneration, as well as Schmincke, who saw calcium concrements following degenerative changes of nervous and glious tissues, and calcification of the vessels and overgrowth of glia, believe that a true inflamma-

FIG. 936.—Rupture of abscess of l. occipital lobe into lateral ventricle (posterior cornua); A, abscess opened, dura attached; the chorioid plexus thickened and infiltrated with pus. The pus contained in the ventricles (int. pyocephalus) was washed out at autopsy. Soldier 21 years old; shell wound. ½ nat. size.

tion has been demonstrated in encephalitis neonatorum (Harbitz). Schwartz believes the condition is a traumatic softening process, caused by differences of pressure occurring during the birth of the child. Hemorrhages and softenings in the pia may be caused by the same conditions. According to Siegmund, the conception "encephalitis congenita" should be abandoned.

Suppurative Encephalitis. Brain Abscess.—A suppurative encephalitis leading to the formation of a brain abscess is related to the entrance of pyogenic organisms. These may (*a*) follow traumata, (*b*) spread from a neighboring part, (*c*) be metastatic via the blood stream from a distant focus.

(*a*) **Traumatic brain abscesses** are often of ichorous character. Bacteria enter the brain through open wounds of the skull, frequently with a foreign body. Abscess formation follows directly. Thus, a few days later, the course taken by a bullet through the brain may be a green yellow, purulent, ragged tunnel throughout its entire length, while, without entrance of bacteria, there is merely red softening. Frequently the primary traumatic abscesses are multiple in one region (with foreign bodies, splinters of bone), and, by confluence, may become an irregular branched abscess (Ernst, Hart). Not rarely, however, bacteria reach the brain from wounds of the soft parts of the skull via the lymph channels. It is important to remember that the external wound may be of minor severity, and may have healed, while the brain abscess gradually becomes larger; in fact, it may not show itself for weeks, months, or even for many (10 to 20) years; the portal of entry may have healed long before. When blunt traumata to the skull, without external injury, are followed by abscesses, it is usually because virulent bacteria present in the air cavities of the skull are mobilized and spread to the brain; see under traumatic meningitis (p. 1828).

(*b*) **Brain abscesses** by continuity are usually from the middle ear (lit. by Korner), in suppurative or fetid otitis media or mastoiditis, as a general rule, from those of long standing duration (even decades) (pp. 1815 and 1829). They are usually solitary, or if there were several abscesses in the beginning, they become one by confluence. The contents are purulent, or thin and watery (for anerobic bacteria, see E. Rist, Chon, Mucha, Muller) and may contain blood coagula and the gases of decomposition in total amount even to 100 ccm. The site of the abscess is always quite close to the diseased ear or the corresponding temporal bone, or, (about 2½ times as rare), in the cerebellum. Usually they are enclosed by a layer of cerebral substance; more rarely, they are in communication with a focus of suppuration in the bone. In children, otitic brain abscesses are more frequent in the cerebrum, in adults, more frequent in the cerebellum. They generally occur more frequently on the right side than on the left. The dura adheres to the surface of the brain over the abscess. The routes by which entrance is gained to the brain are not known with certainty (retrograde lymph—or blood stream?).

Occasionally, a brain abscess follows caries of another bone, or a meningitis, or nasal or accessory sinus disease; in the latter case the abscess is usually in the frontal lobe. (Lit. concerning rhinogenous cerebral abscesses by Hajek; see also Heigel.)

(*c*) **Metastatic brain abscesses** are multiple (over 100), usually small, most numerous in the pallium, less in number in the brain axis or in the medulla oblongata. Isolated abscesses may, however, reach the size of

cherries to walnuts. They are most numerous in pyemic infections; in septic endocarditis, plugs obstruct the vessels and produce first an encephalomalacia, and then abscesses. The left hemisphere is involved much more frequently than the right (see p. 1886), chiefly in the region of the left artery to the Sylvian fossa. Metastatic abscesses, occasionally in large numbers, originate from wounds of distant parts, or softened tumors; they are fairly common in suppurative or ichorous pulmonary and bronchial diseases, among which bronchiectasis may be mentioned, which is more frequent in males, and in which, according to Schorstein, brain

Fig. 937.—Metastatic subcortical abscesses in the brain after suppuration of the posterior mediastinum; woman 25 years old (see text). Frontal section through posterior right central convolution; viewed from in front. Below, the optic tract, and the occulo motor nerve; brain strewn with numerous similar abscesses; $\frac{4}{5}$ nat. size.

abscess is the second most frequent cause of death; tuberculosis of the lungs or empyema of the pleura less commonly gives rise to emboli (p. 1906). Cases are on record in which a large single brain abscess was the only secondary suppuration following operation on distant parts, *e.g.*, ligation of hemorrhoids, or lithotomy. Metastatic abscesses are rare in the infections with fungi or actinomycosis (see the case mentioned on p. 1770).

Many of the so-called idiopathic abscesses are probably of traumatic origin; the portal of entry was unimportant and healed completely. Others are histologically tuberculous, or secondary to a cerebrospinal meningitis, or other infectious disease such as erysipelas, influenza, typhoid fever, which either directly or through involvement of the middle

ear, produced the abscess. The upper nasal passages, as well as the mouth and the pharynx, may be hidden portals of entry. In a 25 year old woman, the author found the point of origin in an occult, old, suppurating bronchial lymph node which had perforated into the esophagus and produced local suppuration and difficulties in swallowing; in the brain, most numerous in the hemispheres of the cerebrum, were multiple rounded abscesses containing thick green evil-smelling contents (fig. 937).

The appearance of the abscess differs according to its age. Fresh suppurative foci are softened, yellow or punctate red spots; as suppurative liquefaction proceeds, they become smooth or ragged, rounded cavities containing light green or sulphur yellow, odorless, or evil-smelling fluid, composed partly of pus and partly of decomposition products in which floating masses of debris are found. Among the bacteria are staphylococci and streptococci, diplococcus pneumoniae, many species of anerobes (see above). Darker green, or black discoloration is caused by sulphide of iron. The neighborhood of the abscess is edematous and softened. If the focus is on the surface, a suppurative meningitis quickly follows. The abscess extends by softening of neighboring tissue, or by confluence of other foci; it may spread in the direction of the fibers of the corona radiata (see the injection experiments of Ahrens who found dye stuffs in the ventricles after injection into the pia; see Ernst), it may reach the lateral ventricles, undermine their walls, jut into their cavities, or rupture into their lumena; this is particularly frequent in otogenic abscesses of the temporal lobes, and in the abscesses following gun shot wounds of the skull. Internal pyocephalus, meningitis or increased intracerebral pressure lead to death.

If the abscess ruptures into a ventricle, it may be followed by meningitis, inasmuch as the pus spreads either from the inferior cornu of the lateral ventricle through the transverse fissure (between hippocampus major and the corpora quadrigemina) or through the fourth ventricle, the normal apertures of which communicate with the subarachnoid space (p. 1804); there then appears a median localized suppurative meningitis which finally spreads to both hemispheres or spinalwards.

An older, larger abscess is usually rounded or egg-shaped, and as a rule solitary, since multiple foci lead to rapid death. According to its situation, it may compromise and flatten the convolutions. In about one month, but sometimes in fourteen days, the abscess becomes encapsulated from the surrounding tissues by a tender white, or gray red membrane composed of granulation tissue.

As in general, the glia plays a minor part, even though in the beginning, as the investigations of Homen show, both connective tissue and glia fibers take part, in its further course, however, when the suppuration is

brought to a standstill, the connective tissue production becomes ascendent while the glia fibers undergo destruction to a small amount of thickening around the tough fibrous membranes. In very old abscesses, (Nauwerck investigated one thirty-eight years old), there may even be a substitution of the mesodermal capsule by ectodermal glious tissue. Similar pictures were seen by Glass in old gun shot wounds (lit. in appendix).

In from two to three months, the membrane is thick, tight, fibrous, richly vascular, and contains fibroblasts externally, while the inner layers still consist of a soft gray granulation tissue which is called a pyogenic

FIG. 938.—Lobed cystic abscess detached from its soft surroundings during autopsy; boy 3½ years old; more details in text; ¾ nat. size.

membrane as long as there is pus present. This capsule later becomes tougher, and the inner surface as smooth as that of a cyst. The encapsulated abscess may occasionally be lifted out in toto, or, at autopsy, it may even fall out of its softened surroundings. The size varies between that of a pea to a fist. Naturally, those in the cerebral hemispheres are largest.

As an example, the author lifted a sac as large as a fist from the right cerebral hemisphere (temporo-occipital lobe); it contained 150 ccm. of thick, cloudy, pale green, mucoid, odorless, bacteria-free fluid. The process occurred in a three year old boy with chronic empyema of the pleura. Acute cerebral symptoms were caused by inflammatory edema, and were apparently spontaneous. (Fig. 938.)

Course. The abscess, particularly if of traumatic origin, (occasionally multiple), may remain stationary and latent for many years without interfering particularly with cerebral functions. The formation of pus

finally ceases, and in older abscesses is, in general, very small in amount. It is yellow green, or tougher and mucoid; if the focus is small, it may even calcify and become brittle and mortar-like. But an older focus, even if surrounded by a fibrous capsule, may grow by progressive suppuration in the capsule itself (details by Homen); it occasionally flares up quite suddenly, particularly under the influence of trauma, but perhaps even spontaneously (see the observation of the author, p. 1906); there then quickly develops an extensive inflammatory edematous zone of softening. In such cases, it is necessary to postulate a surprisingly long, (even decades), functional latence of the bacteria or their toxins. When the pyogenic membrane is extensive, the cause is usually the staphylococcus. When the bacteria die, the healing processes soon cease.

New foci may appear in the neighborhood of an older abscess.

Death occurs almost without exception, in spite of operative help: through fresh infection of the surrounding cerebral substance, a new encephalitis, which kills by edema of the brain and pressure; or following rupture into the meninges or the ventricles. Coma ensues with the symptoms of paralysis of the vagus and of the respiratory centers (Cheyne-Stoke's respiration, etc.); through thrombophlebitis of the sinuses, as in chronic otitis media; pyemia with lung abscesses may be secondary processes, and the actual cause of death.

Spontaneous emptying of an abscess externally into the nasal or tympanic cavities is quite rare.

(Lit. concerning brain abscess by H. Oppenheim, Nothnagels Spez. Path. u. Ther., Vol. IX, part 1, abt. 3; and Ballance. Some points in the Surgery of the Brain, London 1907.)

Chronic Encephalitis. Sclerosis.—(Consult p. 1877.) Many authors discuss progressive paralysis under this heading (p. 1864).

Sclerosis of the brain is fundamentally a primary degenerative or inflammatory destruction of nervous tissues followed by growth of the neuroglia which leads to thickening of this supporting structure. In the relatively early involvements, (periphery of foci), which are soft and gelatinous, there is a fine, soft framework of neuroglia, in the meshes of which are dead or dying, degenerated medullated fibers, myelin droplets, fat droplets, and fat droplet cells (p. 1875); the latter may be so numerous that they produce yellow streaks in the tissues. Lymphocytes appear only as isolated bodies in the glia, the sheaths of the vessels, and their immediate vicinity. The blood vessels are thickened and hyalin, and, especially in the early stages, their walls are particularly rich in nuclei. The older stages are characterized by hardened, elastic foci, which cut like rubber or tough leather; they are waxy, or glassy, red or gray red or pure gray, sometimes pale, almost white. Microscopically, the network

of glia is quite thick, and destroys the nerve fibers, or if the focus is in the gray matter, the ganglion cells; frequently, however, isolated, medullated axis cylinders are preserved. Fat droplet cells are found in small numbers; some lie in the adventitial lymph spaces of the blood vessels. Many authors differentiate a primary sclerosis arising in the neuroglia and leading secondarily to atrophy of the nervous structures, and a secondary sclerosis following primary destruction of the nervous tissues. Schaffer differentiates three kinds of hypertrophy of the glia: (1) primary tumor-like overgrowths, (2) secondary replacement overgrowth, (3) growth following diseases of the glia itself, characterized by rapid destruction of the hypertrophic glia.

In the rare diffuse sclerosis of the brain, a larger part of the organ, occasionally the whole surface of a hemisphere, is involved and takes on the consistency of leather. The gray cortex is much darker and thinner than normal. The surface may be smooth, or atrophic, wrinkled and folded. The pia is thickened but not overgrown (Giannuli, lit.). Occasionally there is hydrocephalus. Not infrequently it is accompanied by idiocy, or by motor irritative symptoms, particularly epilepsy of the encephalitic type. The condition in general is an interstitial chronic encephalitis. Etiologically, there may be considered congenital and acquired lues (spirochetae, see Giannuli), perhaps also alcoholism or meningitis in previous years. There is also a type of diffuse sclerosis of the brain and spinal cord which presents a particular disease complex (Huebner); lit. by Haberfeld and Spieler, who emphasize the pathogenetic unity of diffuse and tuberous cerebral sclerosis. See also Zingerle (lit.), and the histology by Neuburger.

But tuberous (hypertrophic) sclerosis (and gliosis) deserves a special classification (Bourneville, Hartdegen). It occurs mostly in children and younger individuals, (the highest age observed by Neurath was 35 years, Vogt 37 years, but Nieuwenhuijse saw a very rare case at 75 years); there is usually a high degree of idiocy; in a number of cases, however, there were merely minor defects in the mentality, but epileptic manifestations on the motor side (H. Vogt).

Here and there in the cerebral hemispheres are hard, moist, white, glistening or speckled gray, white, regularly disseminated thickenings which broaden single convolutions; or, as in fig. 939, larger complexes of convolutions are involved, or more circumscribed nodules develop, inside of which the border between the cortex and the medulla is wiped out. Moreover, in more than half of the cases, there are hard, irregular, tumor-like prominences, more rarely, larger circumscribed gliomas, in the walls of the lateral ventricles (fig. 949), or, sclerotic linings as thick as one centimeter which reach with tongue-like processes into the bordering

medullary zones with obliteration of the posterior cornu. In a 24 year old baker, the author saw tuberous sclerosis of the cortex of the cerebrum, multiple tumors in the ventricles, innumerable tumors in the kidneys, polyposis of the intestines, and multiple small skin tumors. More rarely the cerebellum or other parts are affected. The tuberous prominences and the tumors in the ventricle may be as large as chestnuts, as in the case illustrated fig. 946, (for histology, see p. 1926); they are composed of glious tissue, in which the nervous structures are diminished, but which contain structures resembling ganglion cells or large glious spindle cells (neuroglia astrocytes). Extensive calcification, particularly of the vessels, in the form of branches, twigs, strands and nodules, is quite common. Because of the ganglion cell-like picture, or because he assumed that true ganglion cells are differentiated, Hartdegen proposed the name "glioma gangliocellulare" which was adopted by Ziegler and others. More recently, however, the ganglion cell nature of these cells has been questioned. Krause believes they are polygonal giant glial cells (see also Jonas). Marchang (Bottcher) believes they are cells like those in true gliomas; he was able to find all transitions from finely branched neuro-glial cells to the colossal ganglion-cell-like structures with very large nuclei; by others, they are said to be abnormally developing cells from an anlage which has not as yet been differentiated. All sorts of transitions exist from diffuse gliosis to occasional true circumscribed gliomatous blastomatous tumors in the ventricles. Thus tuberous sclerosis is an interesting border line condition between malformations and tumors; in these cases it is customary to use the term "tumor-like malformations." It is interesting that there is often a coincidence of (*a*) peculiar multiple, usually benign, mixed tumors (hemangio—lipo—myomas) of the kidney, which, with the exception of a case of Bielschowsky and Gallus, do not lead to enlargement of the organ; (lit., Harbitz). This phenomenon has been described, among others, by Lubarsch and Nuernberg, and by the author in the above mentioned case, fig. 939, and by W. Fischer in six others (fig. 721, p. 1390). Less often, the tumors are malignant, (mxyoliposarcoma, Harbitz), as in the case of de Montet with the character of a hypernephroma. (*b*) They are sometimes combined with multiple rhabdomyomas of the heart (fig. 34, p. 84), as well as (*c*) with adenoma sebaceum disseminatum, or better, naevus sebaceus, (tumors in the face, cheek, see Pringle), and other skin diseases (naevosis; literature concerning the types of these by Jadassohn, Schuster); and occasionally, as in an observation of the author, with pendulous fibromas of the skin (see also Kufs). See van der Hoeve, concerning the finding of tumors in the retina in six cases of tuberous sclerosis. Tuberous sclerosis seems thus to be an endogenous, familiar—hereditary, disease; (see Kirpicznik, H. Berg).

The relationship is frequently pointed out between tuberous sclerosis and general neurofibromatosis, (see under nerves; Bielschowsky and Pick, Orzechowski and Nowicki, Bielschowsky and Gallus). But Nieuwenhuijse opposes this conception; both diseases are different, not only in

FIG. 939.—Tuberous sclerosis, principally of temporal lobes, especially the left, but also numerous other places changed into a hard white more voluminous mass. Leptomeninx pulled off with numerous finer cerebral nerves. There were also several small tumors on the inner surface of the lateral ventricles; boy 7 years old with epilepsy; also had numerous rhabdomyomas of the heart and many small tumors of both kidneys; weight of brain 1250 gms.; ¾ nat. size.

their clinical manifestations, but also in their anatomical changes; those found in the brain in neurofibromatosis (tumors, small glious foci in the cortex and others) have their counterparts in the occasional fibromas found

in tuberous sclerosis. In spite of these objections, Bielschowsky believes they are identical in principle, and are dependent upon congenital disturbances of development in the, as yet, undifferentiated, nervous mother cells, the neuroepithelial cells, which, according to the situation in which they begin to grow, *i.e.*, whether in the central, peripheral or sympathetic nervous system, give rise to tuberous sclerosis, or neurofibromatosis, or a combination of these two conditions; he therefore speaks of "neuroinomatosis centralis, peripherica and universalis."

Lit. concerning tuberous sclerosis by Neurath and H. Vogt, Bundschuh, F. Bohm (lit.), C. S. Freund, Hanser, Mittasch. Other literature under rhabdomyoma of the heart (p. 84) and mixed tumors of the kidneys (p. 1391); see there for illustrations.

See p. 1472 for sclerosis in the so-called hypertrophy of the brain.

In a peculiar disease usually of youthful individuals, which is called multiple disseminated sclerosis (sclerose en plaques), there appear foci in the brain (and isolated cranial nerves) and in the spinal cord (sclerosis cerebrospinalis multiplex), more rarely in one or the other. These are small rounded, or larger, peculiarly angular foci of gray, gray red, pale red or gray yellow color, of hard and tough consistency cutting like rubber, and of a cloudy, glassy appearance. These islands may be somewhat prominent, or at the same level as the surface of the neighborhood, or, more frequently, sunken. Here and there in the foci are small gray white, or yellow, or red stripes which represent vessels. Many foci are of microscopic size. Isolated parts, *e.g.*, the pons or the medulla oblongata, may be almost completely replaced, and thus be irregularly hardened and shrunken. The largest of the lawlessly scattered foci usually lie in the medullary substance of the cerebrum and around the ventricles.

In the spinal cord, the gray blue foci may shine through the soft meninges. The white substance is chiefly the site of the change, but occasionally the entire cross section over some lengths is white gray to gray blue, glassy, transparent, cutting like rubber and markedly shrunken. Immediately above and below this part, the cross section may be macroscopically unchanged, or show but few foci. In general, it is characteristic of the foci not to involve any particular system of fibers. Just as many cerebral nerves (particularly the optic at the chiasm) are involved; parts of the roots of the spinal nerves may also be gray. Even in a more loosely arranged areolar, sieve-like focus, these sclerotic foci microscopically show "naked" swollen, dull, spindle-shaped axis cylinders without medullary sheaths within the thick network of overgrown glia which contains enormous fibers with minor numbers of nuclei, and sheathless nerve fibers (with many granule cells in fresh foci, see Rossle). The persistence of the axis cylinders can be demonstrated by Bielschowsky's

silver method (confirmed by Bartels, Rielschowsky and Redlich), and is believed due to the fact that, even in large sclerotic foci, secondary degenerations are but mild in degree, and do not produce any destruction of the paths of the fibers. The discontinuous destruction of the medullary sheaths with relatively normal axis cylinders is characteristic, and causes the irregular configuration of the foci: their position indicates that there is a certain relationship between them and the blood vessels (Marburg). For the histological details, see Spielmeyer.

The views concerning the histogenesis and etiology of multiple sclerosis are widely divergent. Borst, Goldscheider and Balient believe the process is an inflammation coming from the vascular or perivascular tissues and extending to the medullary sheaths, (destruction of myelin); or, as Borst emphasizes, changes in the vascular system with subsequent disturbances of the lymph circulation, constitute the point of origin of the pathological changes in the nervous system. This is followed by a reactive overgrowth of the glia. The beginning stages therefore represent a multiple acute encephalitis or myelitis. Siemerling and Raecke also believe in inflammatory processes, localized to the areas of vascular supply, which would explain the formation of the foci. Flatau-Koelichen consider that the vessels have been attacked by an unknown toxin, (autointoxication; Ronne and Wimmer speak of metatoxins).

Etiologically, there are considered the above mentioned acute infectious diseases (Marie), but more particularly chronic intoxications with poisons (Oppenheim), carbon monoxide, the puerperium (Balient), and metallic traumata (Mendel, see also Maschmeyer, lit.); the latter is not so easily understood as the action of an infectious or toxic agent, *e.g.*, arising during the puerperium. Siemerling and Raecke emphasize the inflammatory nature of the disease. Syphilis is also held responsible (Catola, Perrin-Parisot). See p. 1919, concerning malarial foci (Durck), which may lead to the symptoms of multiple sclerosis. Among exogenous causes, there is thought to be some relationship with wood ticks, and it is pointed out that there is a preference for individuals working on farms (Dreifuss). The first signs of the disease occasionally appear during pregnancy; repeated pregnancies are often followed by a significant increase in the symptoms (Siemerling). Strumpell, Ed. Muller, and others believe it is caused by an endogenous factor, while Oppenheim, who first showed that many cases may be dated back to earliest childhood, believes that anomalies of development are predisposing, but not essential, causative factors, (lit. by Ed. Muller, Borst). While the numerous attempts of Siemerling and Raecke to produce the disease in animals gave only negative results, Kuhn and Steiner succeeded in obtaining nervous disturbances and paralysis in animals by the injection of the cerebrospinal fluid and blood

of humans with the disease; spirochaetes were found in the blood during life and post mortem and also in the liver; they are delicate and slender and are quite similar to those found in Weil's disease (p. 1019). Siemerling then showed the presence of living spirilla in the sclerotic foci in autopsies done very shortly after death; these were evident in both dark field examinations and in stained preparations. Marinesco, in his experiments with cerebro-spinal fluid and blood, obtained inconstant results, but Simons and Kalberlay reported positive findings. Kuhn and Steiner found foci in the brain (medulla) of an ape which had been inoculated one year previously: the foci were identical with those found in humans; spirochetes were not recovered, however. Rothfeld-Freund-Hornowski (lit.) in their experiments found no significant or specific changes in the central nervous system, and obtained constant negative bacteriological results; they therefore believe the picture in animals is not caused by a virus. The question merits further investigation. As varied as is the distribution of foci, just so inconstant is the clinical picture (Oppenheim). In typical cases there is presented the following symptom complex (Charcot): motor weakness, usually combined with muscular rigidity, spastic, paretic, often staggering gait, intention tremor, nystagmus (rhythmic jerking with turning of the bulbi), partial atrophy of the optic nerve (Uthoff), scanning speech (the words are hesitating and disconnected). These may be accompanied by apoplectic attacks and diminution in the intelligence. The abdominal reflexes disappear quite early (Hedde).

In **paralysis agitans** or Parkinson's disease, a disease of old age, sclerotic processes are found in the central nervous system, chiefly in the posterior and lateral tracts, and as foci around the smaller vessels similar to those in other senile processes. The dignity of this disease described by von Redlich and others is, however, questionable; see Oppenheim, lit. More weight should be laid on changes in the ventricular nucleus, accompanied by symptoms quite similar to Wilson's disease with its paralysis agitans sine agitatione (p. 970; Lewy). See page 542, concerning the possible relations to the parathyroid glands.

In alcoholics, Marchiafava and Bignami, Rossi, found localized sclerotic foci in the corpus callosum and in the anterior commissures, which were secondary to degenerations (see Marchiafava, Bignami, Nazari, lit.). Sarteschi and Cesaris-Demel also saw gray degeneration separated from the cortical gray matter by white stripes and attacking the periphery of the centrum ovale and radiating out into the white substance of the convolutions. Similar markings can be found in normal brains hardened in formaldehyde, so that in the brains of alcoholics there is most likely a preference displayed by the damaging toxin for this zone.

Infectious Granulation Tumors of the Brain

Tuberculosis of the Cerebrum.—The occurrence of small miliary tubercles in the cerebral substance as an accompaniment of tuberculous meningitis has been mentioned on p. 1832. Occasionally, larger tubercles of the cerebrum arise in this manner; more rarely there are larger numbers in all parts of the brain. More frequently these solitary, or, better, conglomerate tubercles, are independent of disease of the meninges, but develop slowly after tubercle bacilli have been distributed via the blood stream into the cerebral substance.

Conglomerate tubercles are usually rounded, spherical, or nodular bodies which may be solitary, but, as a rule, are multiple. In the cortex, they are occasionally irregular and angular, similar to gummata. They reach the size of peas, hen's eggs or apples (fig. 941). Clinically they produce the symptoms of tumor (p. 1937).

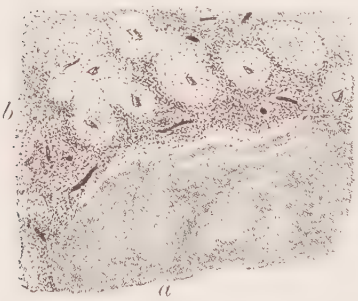


Fig. 940.—Marginal area of solitary conglomerate tubercle of brain of 3 year old child. a, homogenous caseous center; in the periphery (b) granulation tissue with blood vessels and tubercles (light) with giant cells (dark areas in tubercles). (Low power.)

Older nodules are caseous, of light green color and fairly firm. The caseous masses on section are frequently irregularly striped in various directions, or show here and there a hint of concentric lamination. As long as the nodule does not enlarge, it appears as a red gray or gray, soft, relatively small zone of granulation tissue, in which there are many miliary and conglomerate tubercles (resorption tubercle) containing numerous bacilli. In this zone, which gradually blends with the cerebral substance, the tuberculous granulation tissue progresses. In very late stages, the zone of tuberculous granulation tissue is no longer present, but has been changed into fibrillar connective tissue, which surrounds the totally destroyed conglomerate tubercles, and is externally fairly sharply separated from the neighboring sclerotic or softened cerebral substance; this is the reason for the easy enucleation of the tubercle at autopsy. Bacilli cannot always be demonstrated tinctorially in later stages.

The older nodules may easily be lifted out in toto, but if there is considerable softening in the neighborhood, the conglomerate tubercle may, like many sarcomas, drop out on mere manipulation.

Abscesses containing caseous or yellow green pus are produced from softening and liquefaction of the tuberculous nodules. The wall must then be examined for tuberculous characteristics.

Large conglomerate tubercles are found most frequently in younger individuals, particularly in children with tuberculosis of the lymph nodes (see p. 255 and following). They may enlarge rapidly or remain stationary for long periods of time. In addition to the conglomerate cerebral tubercle, older individuals may have merely negligible foci in the rest of the body; thus, for example, in a 51 year old woman, the author found a tubercle in the cerebellum as large as an almond, old scars in the lungs, and chalky bronchial lymph nodes. Preferential sites are in the basal parts of the brain, the cerebellum, particularly the upper convolution pons and the basal parts of the cortex of the cerebrum. The

FIG. 941.—Very large conglomerate tubercle in right cerebellar lobe and pons of girl 6 years old; oblique horizontal section. Circumference of skull 52 cm.; contents of ventricles 400 cc. clear fluid; increased cerebral pressure; diastasis of coronary suture, choked disk; portions of cerebellum forced into foramen magnum; lumbar puncture without result; cerebellar symptoms for 18 mos.; P, pons; D, dura; Ab, basilar art.; IV Vent,—fourth ventricle; ¾ nat. size.

preference displayed for the cerebellum (see fig. 941) accounts for the frequent compression of the aqueduct of Sylvius, obstruction to the outflow of the ventricular fluid and marked hydrops of the ventricles. The increased brain pressure may lead to dilatation of the child's skull (open fontanelles and sutures). Local or extensive tuberculosis of the meninges may follow.

Syphilis of the Brain and the Soft Meninges.—Most of the anatomically specific changes occurring from syphilis in the brain are spread from the meninges. This applies to cerebral syphilis in contrast to the changes in the brain occurring in progressive paralysis (p. 1864), which likewise is caused by spirochetae. It is still unknown why in one case there should be paralysis, and in another case cerebral lues.

The lesion produced by cerebral syphilis is a basal, gummatous meningitis. This is composed of circumscribed solitary or multiple inflammatory foci, or of a more diffuse specific tissue reaction in the meninges. In the beginning, the gummatous tissue is spongy, colloid, or richly cellular; it then undergoes caseation and later becomes fibrous. It is quite delicate and may undergo total caseation; more frequently, however, it becomes caseous in spots (yellow), while in other places it is bacony (gray), in other places fibrous (white), and in still other places young and fresh. The infiltrating gummatous tissue leads to tight adhesions between the meninges and the cerebrum, particularly around the Sylvian fossa; later it invades the brain substance itself. In severe cases, a thick opaque membrane covers the basal structures, or extends as a thick veil over the cerebral nerves and blood vessels.

Occasionally the changes are localized to the Sylvian fossa and cannot be found at first glance on the base of the brain. (Therefore it is best to uncover the Sylvian fossa at every autopsy.)

Exceptionally, a syphilitic meningitis covers the convexity; (see Cronquist).

The gummatous new growth extends to the epineurium of the cranial nerves and infiltrates the adventitia of the blood vessels. The nerves, particularly the optic and the oculomotor, are therefore thickened and brought to atrophy, and clinically, there are paralyses or pareses of isolated ocular muscles, particularly those supplied by the oculomotor nerve or its branches. The changes about the arteries are mostly in the form of syphilitic periarteritis, mesarteritis, and endarteritis, and may be accompanied by gummatous necroses. (For more details, see under arteries, p. 129, and see Heubner, Nonne, Luce, lit., Staemmler.) This of tenleads to obstruction of a vessel, particularly when thrombosis is added, with subsequent multiple focal, smaller or larger, non-specific necroses, usually white, but perhaps red. Obstruction of the artery to the Sylvian fossa is relatively frequent and is followed by hemiplegia.

It must again be emphasized that an arteritis of syphilitic nature may be circumscribed to one vessel or system of vessels, thus, for example, the basal, and be the only syphilitic change in the brain. Hyalinization of the smaller cerebral branches with subsequent small foci of softening is relatively rare, but has been observed even in the earliest stages of syphilis (Stursberg).

When acute and subacute syphilitic meningitis occurs, it develops almost without exception in the early stages of syphilis (the first year), and according to Oppenheim, Nonne and others, is quite common; its course, which is usually accompanied by fever, is occasionally particularly malignant (Krause). According to Pirila, a gray yellow exudate collects,

particularly in the region of the chiasm; microscopically, lymphocytic infiltration is predominant as contrasted to tuberculous meningitis in which leucocytes are more numerous (Beitzke, Forster); they accumulate in the neighborhood of the vessels, which show overgrowth of the intima, while the gummatous infiltration regresses. Pirila found many spirochetae in such cases, particularly in the walls of the larger basal vessels. Fahr found extraordinary numbers of spirochetes in a syphilitic gummatous meningitis ending fatally nine weeks after the infection; Ranke, in the speckled cloudy pia (Schmeisser), and Eliassow (lit.), in meningitis gummosa in a newborn, also found innumerable spirochetae. Even in nodular syphilitic meningitis, which may be quite similar to a tuberculous process (see illustrations by Beitzke), there are early gummas produced, usually in younger individuals with fresh syphilis (Verse, Durck, Sugi, lit.).

Larger circumscribed gummata, as a rule, originate from a gummatous meningitis. The overgrowth extends from the membranes in continuo as a multiple, nodular, tumor-like affair in the cerebral substance. The nodules may reach the size of beans. They prefer the base, but occasionally, multiple large gummata begin in the cerebral substance itself, as for example, in the central ganglia. Otherwise those situations injured most easily are predisposed (Virchow). The cerebellum, in contrast to tuberculosis, is seldom involved.

The gummata vary according to their age; early examples are gray red, soft, colloid, or softened in their centers, white in their peripheries, gray red and colloid. Later they become tougher, appear like pale meat on section, or are speckled with irregular, map-like, confluent, angular, yellow, dry, elastic, caseous masses. Sclerotic fibrous changes and caseation occur hand in hand; rarely, as in solitary tubercles, there is total caseation of the entire nodule; more often, the centers are caseous, while in the peripheries are broad zones of glistening gray to gray red color which gradually are lost in the surroundings. Encapsulation, as occurs in older solitary tubercles, is rare. The surrounding cerebral substance may be softened over a broad zone, but may also be mildly sclerotic. Where a gumma is established in the soft meninges, there is a tendency to adhesions to the dura.

Microscopically, the gray places show short, small, spindle cells and varied round cells, such as lymphocytes and plasma cells, eosinophiles; numerous epithelioid cells appear granular, and cloudy, with poorly stained nuclei, in contrast to the epithelioid cells in tuberculosis. Not rarely, there are isolated Langhan's giant cells; exceptionally, many are present. The caseous masses have a granular background of particles of destroyed nuclei, and fat granule cells (see illustrations under muscles).

Miliary non-gummatous necroses of the cortex have been described by Herschmann.

An obliterating endarteritis of the basal vessels (p. 130) or a true gummatous arteritis without gummatous meningitis, also occasionally appears in syphilitics. Its greatest danger lies in the subsequent closure of the vessels and the consequent softening of the brain. Thrombosis of a gummatous basal artery has led to sudden death (lit. by Saathoff). This is called the arteritic form of cerebral syphilis (Nonne, K. Krause, Freund). In children, it is said to be an evidence of congenital lues (Takahashi).

A syphilitic cerebrospinal phlebitis is quite rare when the artery is intact; thick, yellow, striped infiltrates surround the veins; partial thrombosis may follow. For more details, see Verse.

Syphilitic diseases of the brain occur most frequently in the first year after infection, or earlier, (early lues, see p. 1916). Thus, for example, in a case of Pette, infection occurred four months before; in the case mentioned on p. 1917 in a 25 year old man, it occurred 9 weeks after infection; in three cases of Pirila, the individuals died from luetic meningitis eight to ten months after infection in spite of salvarsan and other specific treatment. Early luetic meningitis seems to be more common since the use of salvarsan (Pette). It is rarely seen 10 years after infection (Naunyn, Oppenheim). Lues of the cerebrum may be the only manifestation of syphilis found at autopsy.

For the differential diagnosis between tubercles and gummata in general, see under muscles.

Tubercles are mostly deep and more spherical, more sharply outlined from the neighborhood, usually caseous in toto, green in color, with a gray translucent capsule of granulation tissue in which macroscopic miliary tubercles are often visible; they are avascular, occasionally purulent, usually much larger, and are particularly frequent in children. The finding of tubercle bacilli is pathognomonic. The findings in other organs can be considered. Miliary nodules should be sought in the soft meninges in the neighborhood and in the dura.

Gummata are usually superficial, in connection with the meninges, more rarely isolated in the depths, are frequently angular, irregular, circumscribed or flat, and, as a rule, less sharply outlined from the neighborhood. They are frequently bacony, partially yellow and caseous, surrounded by a broad area of granulation tissue in which there are no caseous nodules, as is so frequent in tuberculosis. They have less tendency to softening, although it does occur, and leads to the formation of cysts; they are more frequently small, and are more often found in adults. Occasionally spirochetae may be demonstrated in early stages in the edges of fresh gummatous foci.

If the specific causes cannot be found, the differentiation between gummata and tubercles is sometimes entirely impossible (Nonne).

Gummata may be differentiated from tumors through their larger caseous areas, their less distinct surfaces, the presence of less cellular elements, and the pathological appearance of the cells and nuclei. In one case, Goldzieher (lit.) found enormous masses of plasma cells which never occur in tumors.

Actinomycosis is rare, almost always secondary, and usually in the form of an abscess, but occasionally as a granulation tumor. The conditions under which actinomycosis of the brain appears are the same as those mentioned under basal purulent actinomycotic meningitis (p. 1837), *i.e.*, extension via the blood stream. Primary actinomycosis, as in the case of Bollinger, is quite rare; he describes an acute granulation tumor as large as a hazel nut in the third ventricle, with fatal result. Lit. by Geymuller, Belkowski, Corinth, Sagredo. The collection at Basle contains a preparation showing actinomycotic abscesses in the left crus cerebri ad pontem from a 34 year old woman with actinomycosis of the lower jaw, hyoid bone, the retropharyngeal tissue, the base of the skull, the dura and the soft meninges. Consult the observation of the author described on p. 1770.

Leprosy is very rare (lit. by Stahlberg).

In **pernicious comatose malaria** or tropical malaria, Durck found microscopic foci of glia cell growths which were particularly pronounced around the capillaries (see also Oesterlin), with accessory cell proliferations around the ganglion cells, often in combination with neurophagia. Also, among other things, he found peculiar cellular nodules particularly numerous in the cases which—apart from the smoke gray discoloration of the brain following the embedding of many plasmodia and malarial pigments in the capillaries—were characterized by punctiform hemorrhages. These may also, however, be found in most of the cases of fatal malaria. Their number is varied, they appear in the medulla of the cerebrum and never in the cortex, and resemble fresh foci of multiple sclerosis (disappearance of medullary sheaths, glial cell overgrowth, retained axis cylinders); the proliferating cells of these nodules come from glia cell overgrowth. In the center of a nodule there is usually a capillary filled with plasmodia. The elongated spindle and oval cells are arranged radially from the necrotic centers (Spielmeyer; "daisy figures"). These nodules, which seem to represent a specific reaction of the nervous tissue against the irritation of intravascular malarial parasites, Durck classifies with the infectious granulomas, and proposes the name "malarial granuloma." See also Weingartner, who described calcification in damaged parts of the brain, (Seifarth).

Malarial granulomas have no histological similarities to the nodular structures in the brain in spotted fever (pages 122, 781 and 1878), which prefer the cortex of the cerebrum and are composed of mesodermal and hematogenous cells, according to Durck; they are, however, quite similar to the nodular cell overgrowth in the cerebrum in Chaga's disease (see pp. 212, 524).

Tumors of the Brain

Tumors Which are Specific to the Central Nervous Apparatus and the Retina. —Many authors have emphasized the difficulties of proving the relationship of gliomas of the retina to true gliomas, and their similar ectodermal origins (Wintersteiner, Freeff, Axenfeld). Gliomas of the retina appear only during childhood, are congenital, quite frequently bilateral, and have a familial, inheritable tendency; because of their richly cellular content, they have often been classed with the round cell sarcomas of perivascular arrangement. It is probable that they have an embryonal origin; they grow very rapidly, invade the eyeball, metastasize by way of the optic nerve into the brain, or rupture through the sclera; are always malignant, produce marked changes by their local spread, and give rise to extensive distant metastases, even into the skeleton (Roman); other malformations of the eye are not infrequently coincident (Bergmeister). Wintersteiner proposes the name "neuroepitheliomas" for such gliomas in which there are epithelial-like cells arranged in alveoli or rosettes; he believes they originate from inclusions of cells of the external stratum granulosum and are similar to rods and cones which have remained in an immature state of development. Steinhaus, on the other hand, believes these epithelial cells are enclosed "primitive epithelium" situated in the retina (see p. 1925), which arrange themselves into hollow spheres (the rosettes), and from which, in their further development, there may appear glia as well as nerve cells. Greef, who, in addition to glia cells, describes ganglion cells within them, calls them "neuroglioma ganglionare." Wehrli believes the central parts of some of the rosettes are vessels with degenerated walls, others, epithelial tubes. B. Fischer calls them "neuroblastomas," *i.e.*, tumors which arise from anlage cells of the nervous substance, (primitive neuroblasts), and believes they are similar in all respects, both histologically and biologically, to the neuroblastomas of the sympathetics, *i.e.*, of the adrenals (see p. 1288). The tumor called by v. Hippel, "angiomatosis of the retina," or "angiogliomatosis," is believed by B. Fischer (lit.) to be a mature, benign condition arising in middle or later life, similar in these respects to the comparable tumors of the sympathetics (adrenals); but the ripening of these retinal tumors does not proceed as does that of the adrenal tumors, namely, to the formation of ganglion

cells (see ganglioneuromas, p. 1288), but does to the formation of glia cells. Gliomas have also been observed in the nerve roots of the brain (particularly the acoustic nerve), in the nerves of the spinal cord, in the spinal cord itself, as well as in peripheral nerves. Gliomas of the nose are very rare; in the case of M. B. Schmidt (lit.), it was an external tumor above the point of the nose. Schmidt hypothecates a relationship to an encephalocele; the latter occurred in a case of v. Meyer with the formation of an intranasal solid prolongation of glia. Peterer described a glioma of the tongue, Askanazy, gliomas of the lungs, which he believed were carried there through the hematogenous route in a hemicephalic. See Haberfeld (lit.), for glioma of the posterior lobe of the hypophysis. Gliomatous, or merely glious, parts in teratomas are quite common (see pp. 1585, 1506). See p. 1288 concerning the tumors formerly called gliomas of the adrenals.

Gliomas are the most frequent tumors of the brain, and apart from a very fine connective tissue supporting structure (mesenchymal) and blood vessels, are composed of overgrown neuroglia, the specific (ectodermal) supporting structure of the central nervous system.

The entire nervous tissue is of ectodermal origin, and arises from the embryonal medullary tube: this is composed of primordial embryonal epithelial cells, (glianeurocytes, Held), which differentiate into neuroblasts, (true nervous elements), the ganglion cells, and from these appear nerve fibers, and the arrow shaped slender spongioblasts which are the mother cells of the supporting structure, the neuroglia. According to Broman, the primordially similar epithelial cells differentiate into: I. supporting cells which become (*a*) ependyma cells, and (*b*) neuroglia cells; II. neuroblasts or true nerve cells. The neuroglia is composed of glia cells and glia fibers, both being products of differentiation of the spongioblastic syncytium. There are two kinds of glia cells. The ependyma cells (*a*) retain their primordial embryonal epithelial character and take on a radial arrangement; in fact, they are the bodies of spongioblasts which lie close together. Later, when they line the ventricles and the central canal as a strongly staining cylindrical cell layer, they come into contact with the remaining supporting structure of the neuroglia through basal prolongations which sink deep into the underlying tissue. (*b*) The stellate or spindle shaped neuroglia cells, astrocytes, Deiter's cells, have rounded or polymorphous nuclei and cell bodies difficult to recognize; some have long prolongations and some have short prolongations (Golgi's method). On the basis of studies with his own glia stain—which, however, stains only the fibers—Weigert considered the fibers closely related to the cell bodies, but this idea is no longer recognized (consult pp. 1876 and 1877).

We cannot discuss the question of whether, in the formation of the supporting structure of the central nervous system, there is a mesodermal part—mesoglia, in addition to the ectodermal glia, but see Robertson, Scaffidi.

The microscopic picture of the glioma varies. If we begin with the description as indicated on p. 1877—that it is a nucleated syncytial protoplasmic layer in which fibers are embedded—we see, in general, the same structure in gliomas as in the so-called gliosarcomas, which are merely richly cellular gliomas. But the relative masses of the different structures vary in high degree; in one case, there is a richly fibrous, poorly nucleated

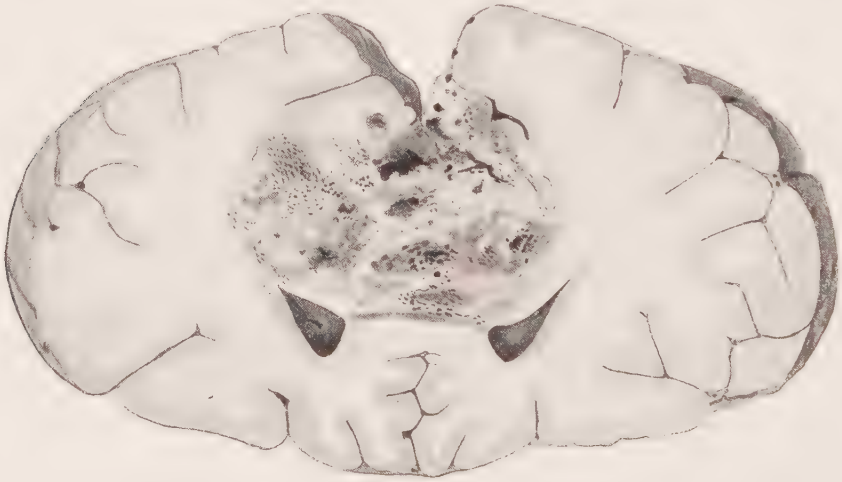


FIG. 942.—Vascular glioma of brain with hemorrhages, necrosis, fatty degeneration, softening, gradually involving normal tissue; frontal section through frontal region. Tumor in the corpus callosum extending bilaterally into the ant. cornua of lateral ventricles and into both hemispheres; man 46 years old; $\frac{4}{5}$ nat. size.

structure; in other cases, a highly cellular and poorly fibrillar tumor. Occasionally, a well defined glious protoplasm can be demonstrated; again, the fibers are very poorly developed; on the other hand, there are cases in which the picture resembles that of primordial glia, *i.e.*, an almost purely cellular structure composed of branched anastomosing masses of protoplasm; and in still others, the cells resemble much earlier forms such as spongioblasts and ependyma cells. There is, moreover, quite a variation in the amount of fibrous (mesenchym) interstitial tissue as well as in the number of blood vessels. The latter are often pathologically formed and frequently show degenerative changes. Glia fibers as well as arrow-shaped spongioblasts or ependyma cells are often attached externally to the blood vessels, and appear as arc-like bundles against the surrounding tissue (fig. 948).

Thus there may be differentiated:

Relatively Poorly Cellular, Richly Fibrous Gliomas (fig. 943).—In imitation of the normal glia, there is a syncytium with more or less numerous nuclei. The latter are of various shapes, but principally small and rounded with little surrounding protoplasm; others show well differentiated protoplasm (glia cells). These glia cells have prolongations which interlace with the more weakly stained, tender fibrillar glia reticulum which is a modification of the cell protoplasm. Furthermore, there are more sharply differentiated fibers which are embedded in the mesh-like protoplasmic syncytium; these fibers which appear through a gradual

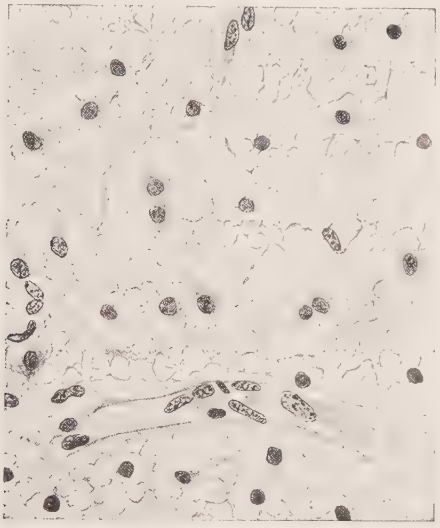


Fig. 943.—Glioma, poorly cellular, rich in fibres of frontal brain of man 20 years old. Iron hematoxylon—van Gieson stain. (High power.)

differentiation in their substance (see below), may, however, also be lacking. The harder the glioma, the more richly fibrous it is.

For staining, the iron hematoxylin method of van Gieson can be recommended; the neuroglia with the protoplasm around the nuclei and the protoplasmic reticulum, the fibers of which are peculiarly compressed and kinked, are stained yellow, the nuclei become brown, the connective tissue (collagenous mesenchymal tissue) is stained red. Finer differentiation between the fibers and the glious protoplasm and the reticulum can be accomplished by more complex staining methods such as those of v. Fieandt, Mallory and others; see also p. 1877.

In many gliomas, there are numerous glia cells with prolongations, the so-called spindle cells or astrocytes, varying in size and in number of prolongations, so that we may speak of cells with long or short prolongations, or of giant cells. These prolongations compose a tangled network

between the cells; but sharply outlined cells are not present in the meshwork. Gliomas particularly rich in such pictures are called astromas (v. Lenhossek) or astrocytomas.

The apparent differences between those gliomas structurally similar in principle to the normal glia, and those astrocytomas—first studied carefully with Mallory's stain by Stroebe—have been explained on the basis of Weigert's neuroglia stain (Storch, and see also Bonome) in this manner: that in the gliomas composed chiefly of large glial cells with prolongations, the chemical differentiation of the interstitial substance has not as yet

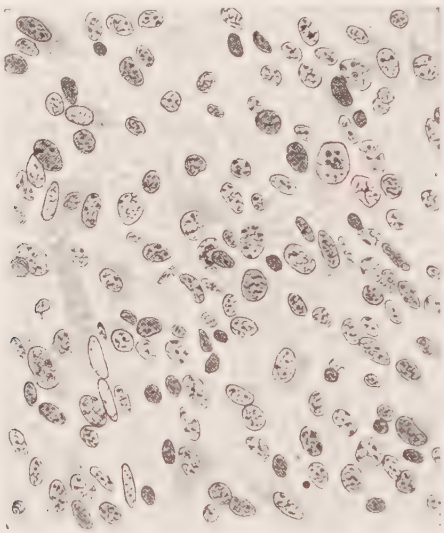


Fig. 944.—Cellular soft glioma of temporal lobe of woman 34 years old; fine glia reticulum, but few thick fibres; same stain as fig. 943. (High power.)

been completed because of rapid cell division; while in the normal glia, which does not contain spindle cells, the glia fibers (the interstitial substances) are differentiated from the protoplasmic-poor glia cells; Weigert therefore assumed there is a complete independence of the fibers from the cells. This hypothesis, however, is now doubted; as Stumpf (lit.) described, the fibers form within the embryological glia cell, but eventually become free of its protoplasm, to return, however, into the protoplasm of another cell; they thus become a supporting substance for the cells, and represent, therefore, a modification of the cell protoplasm, the glia reticulum.

In **richly cellular gliomas** with regression of the fibers (figs. 944–945), the nuclei vary in size; they are often larger than in the first type, are rounded, egg-shaped, or broad and elongated, or entirely polymorphous. Some of the glia cells contain little protoplasm, so that merely the nuclei

lie between the meshes of the glia; some however, have quite a large protoplasmic body. The nuclei, which are often compressed, are rich in chromatin, and may show mitotic figures, as well as direct division, which is foreign to the normal glia. The glious fibrous syncytium may regress more or less, so that the tumor comes to have an appearance quite similar to that of an ordinary round or plump spindle cell sarcoma. When very large, multipolar, often many nucleated giant cells are present, they may, like similar cells in tuberous sclerosis (p. 1908), be mistaken for ganglion cells. Tumors in which the giant cells determine the character of the growth are called giant cell gliomas (O. Meyer, Kraus).

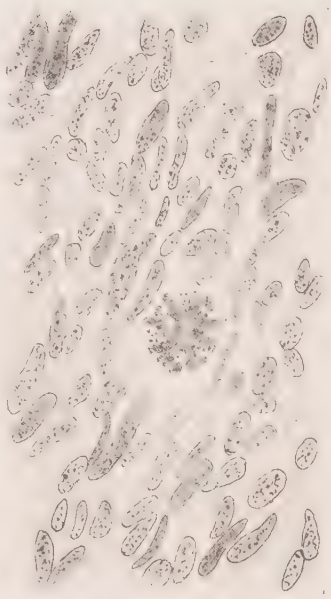


Fig. 945.—Cellular glioma of white matter of right hemisphere; many larger elongated nuclei; large stellate karyokinetic figures; delicate protoplasm; finest glia fibrillae. Same stain as fig. 943. (High power.)

There have been repeatedly described gliomas in which certain cells take on an epithelial character in single layers like ependyma epithelium, or in many layers as in the embryonal neural tube, forming canals, tubes, cysts, alveoli or rosettes. To understand this picture it must be remembered that the supporting tissue of the central nervous system comes from epithelium (p. 1921), which, in certain situations, such as around the central canal and the ependyma epithelium of the ventricles, retains its original character, while the greater part of it develops further into the supporting substance of the nervous tissues. There also occur pure, or almost pure, epithelial and gliomatous tumors containing epithelial structures (see p. 1842), which arise from the walls of the ventricles. Ribbert proposes the name "spongioblastoma" for all those tumors of composite structure containing rosettes and epithelial inclusions of all kinds as far as they are derived from the spongioblasts; when they display the very early stages of development, they are spoken of as "spongioneuroblastomas," because these latter, he believes, originate from the predecessors of the spongioblasts, (the primitive anlage cells of the brain have not as yet differentiated into spongio- and neuroblasts). In such cases, Ribbert also saw black, bile-pigmented, mosaic-like retinal epithelium. Tumors composed of more or less completely mature glia he calls glioblastomas or gliomas, in which the abundance of cells is a sign of an early developmental disturbance, and abundance of fibers a sign of later

developmental disturbances. The author, Muthmann, and Sauerbeck (lit.), described a richly cellular tumor resembling a perivascular sarcoma arising from the ependyma of the fourth ventricle (see fig. 920); these represented intermediate stages between the pure epithelial tumors of the nervous system and the astromas, inasmuch as the tumors, apart from the blood vessels and sparse connective tissue bands, were made up almost exclusively of spongioblast-like, so-called arrow cells many of which radiated from vessels. These arise during the normal formation of the glia, because the epithelial cells retain a long prolongation of their basal side. If such prolongations are subdivided, there arises the actual glia cell, the astrocyte; a histogenetically similar case is described by Roman as neuroepithelioma gliomatosum (p. 1842). Saxer, Bittorf, Borst and

Fig. 946.—Epithelial formations in a glioma (spongioblastoma) of brain (Ribbert, V.A. 225). (Low power.)

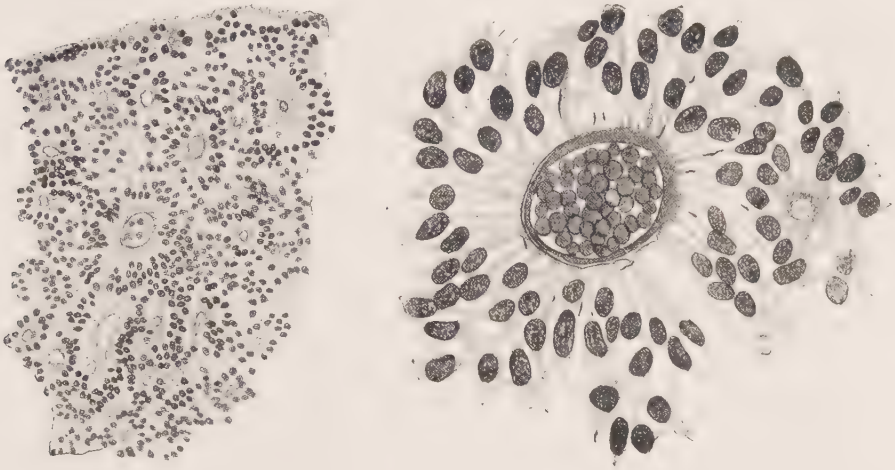
others believe it is possible that astrocytes, when they form tumors, may return to their epithelial form, an hypothesis which makes unnecessary the assumption that there is a germinal inclusion with the presence of epithelial formation; Hart, Ribbert, Landau (lit.), do not believe, however, that there can be such a regression of fully differentiated glial elements and they are probably correct.

Other richly cellular gliomas, such as those seen by the author in the lateral ventricles in tuberous sclerosis, reaching the size of chestnuts, may be composed of long, small cells resembling spongioblasts without the broad ends (*i.e.*, epithelial). The cells may be arranged parallel in bundles and strands, or may radiate from blood vessels and connective tissue bands; parts show transition to smaller thicker cells of irregular epithelial appearance, and other parts to long astrocytes lying in finely fibrous masses; those parts containing but few fine fibers may appear as poorly differentiated strands between the thickly compressed cell bundles. The less fine the fibers that are present, the greater the similarity to sarcomas.

See Kraus concerning a pigmented glioma (giant glial cells with peculiar granulation and pigment).

The richly cellular gliomas which are not always easy to distinguish from sarcomas may also be called "glioma molle or medullare," in contrast to the poorly cellular, richly fibrous "glioma durum." There are combinations of both, and not every glioma medullare is soft. Other gliomas show a gelatinous composition in their wide meshed intercellular substance and thus are called myxogliomas. Others contain many wide blood vessels (teleangiectatic glioma) and show a tendency to marked destructive hemorrhages (the so-called hemorrhagic glioma or apoplectic glioma); see fig. 942.

Sandy bodies are occasionally formed in gliomas (psammogliomas). Medullary gliomas (page 1924) which have also been called "glio-



FIGS. 947 and 948.—From gliomatous neuroepithelioma of third ventricle; blood vessels surrounded by epithelium with radiating prolongations. Fig. to right shows a corona of rays; suggestion of glia fibres in protoplasm prolongations and around blood vessel; Heidenhain stain. (Roman, V.A., 211, 1913.)

sarcomas" (Ewing uses this term; Ernst in Aschoff's text books advises that it be dropped), or (Borst, in the text book of Aschoff), "glioma-sarcomatodes" or "glioplastic sarcoma," are occasionally able to invade the neighborhood (meninges, bones), and also to produce metastases (Schmincke), while poorly cellular gliomas grow slowly and seldom extend beyond the area of the cerebral substance, except into the soft meninges (fig. 951), which they occasionally invade diffusely (Spiller, lit., and see, *e.g.*, the case of Strauss; see also Hildebrandt). Durante calls them pure sarcomas, and Scaffidi assumes that some of them belong to the "mesogliomas," (consult p. 1922). Entirely different structures are found when the blood vessels of a glioma are surrounded by a sarcomatous collar; these are combinations of gliomas and sarcomas. Ewing doubts their occurrence.

The nomenclature is quite confusing. The author does not use the term gliosarcoma, but speaks of richly cellular gliomas. The conception "sarcoma" or "sarcomatodes" leads to obscurity. Sarcoma is a mesenchymal tumor and the epithelial glioma can never be such. The similarity to a sarcoma consists merely in the abundance of cells and in the occasional rapid agressive growth. The mode of spread at the periphery is different in these gliomas than in sarcomas (p. 1933). But not every richly cellular glioma is *eo ipso* malignant; arrow cell gliomas with many cells, as *e.g.*, in the ventricles, may remain local in spite of their large size, and, on the

FIG. 949.—Glioma in dilated l. lateral ventricle in tuberous sclerosis. At a, a light broad sclerotic convolution. (Most of the tuberous sclerotic convolutions were on the outer surface of the hemispheres.) From an epileptic idiot 16 years old with sebaceous adenomata in face; death during epileptic attack; weight of whole brain 1380 gms. Nearly median sagittal section; ⅔ nat. size.

other hand, poorly cellular tumors may, even though rarely, invade the meninges (see fig. 951).

Gliomas originate only from the brain, usually from the medullary substance of the cerebral hemispheres, but not rarely also from the upper part of the vermiform convolution of the cerebellum. They are usually solitary (for exceptions, see Froehlich), roughly spherical, and their consistency and color contrast with the surrounding tissue in which the tumor is gradually lost without sharp boundaries and with no compression (circumscribed form), or the boundary line is entirely indistinct, and the tumor infiltrates its surroundings (diffuse form), so that some of the convolutions or even whole complexes of convolutions become voluminous and relatively soft and flat; even the entire pons, the optic thalamus, etc.,

occasionally become gliomatous (diffuse infiltration, Henrich, lit.), and thus voluminous. Nervous elements, nerve fibers and cells have usually disappeared from the center of gliomas as a point of differentiation from the glioses which are hyperplasias of the glia (see under spinal cord, p. 1979), while in the peripheral parts of the tumor they may be partly preserved. Intranasal gliomas are quite rare; they usually arise from the olfactory nerve or from an encephalocele (Schmidt). Gliomas of the brain usually grow slowly, remain as a rule localized to the parts involved, but may invade the soft meninges. The latter are then opaque and

FIG. 950.—Cystic cavernous glioma in posterior part left temporal lobe; swelling of medulla of l. hemisphere; below the corp. call. (in splenium) the corpora quadrigemini in midline; the aqueduct of Sylvius and pons; boy 9 years old; weight of brain 1650 gms.; ¾ nat. size.

adherent to the cortex. In such a case (53 year old man, glioma in the left temporal lobe), the author saw multiple microscopic ruptures through the pia into the subarachnoid space. Diffuse spread into the meninges of the brain and the spinal cord is quite rare (Lowenberg, lit.). Occasionally gliomas reach the size of a fist. The consistency varies considerably; some are hard, some are soft. The color is yellow white or gray red, but usually more transparent than the normal cortex. As a result of regressive changes (Landau) such as fatty degeneration, multiple smaller hemorrhages, edematous colloid changes, necrosis, etc., the color is often variegated from cloudy white to yellow to gray red, red, brown and purple.

Softening with pseudo fluctuation and false cysts surrounded by tumor tissue is frequent (cystic or cavernous glioma); this is probably caused by circulatory disturbances, for the vessel walls have abnormal structures (Beriel). Sharply outlined gliomas, or gliomas free in a ventricle are much rarer (fig. 949); they are more common in tuberous sclerosis (fig. 949); smaller, usually hard, poorly cellular gliomas are relatively frequent in the lateral ventricles.

Microscopic true cysts, lined by epithelium in the form of rosettes are sometimes found (Stroebe, Beriel).

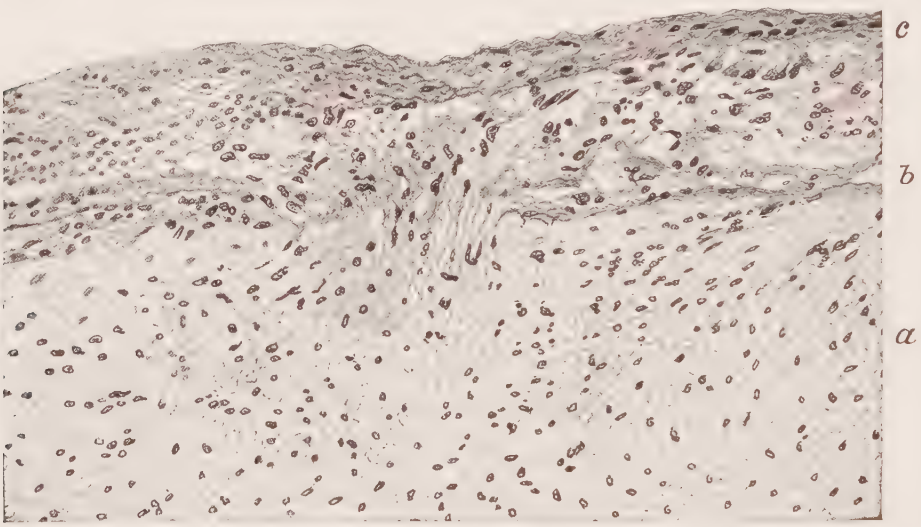


Fig. 951.—Invasion of a glioma into the soft meninges; a, cortex completely replaced by tumor which passes through the boundary to pia (b), filling the subarachnoid space and infiltrating the arachnoid (c). From a tumor of temporal lobe, man 53 years old. (Med. power.)

Cystic changes and fresh hemorrhages may completely alter the character of the tumor and render it unrecognizable to the inexperienced. Extensive fatty degeneration produces a cloudy picture similar to that of white softening. Larger hemorrhages are common in telangiectatic gliomas; they produce rapid swelling, but the tumor itself may be almost completely destroyed, so that at autopsy it seems as though the condition were an ordinary cerebral hemorrhage. The periphery should be examined for isolated foci of tumor tissue which may be present in surprisingly small amounts.

The anlage of gliomas is probably laid down quite early in embryonal life (consult the description of a neuroglioma in a chick embryo described by Podmaniczky). A developmental disturbance probably occurs, which, in the case of circumscribed gliomas, consists of a tissue inclusion, or in the case of diffuse gliomas (Landau), of a congenital malformation,

or a failure of differentiation on the part of the neuroglia. Ernst has described dystopias in the cerebellum which appeared similar to beginning gliomas. In animals, Schaper and Cohen call attention to "indifferent zones" or "epithelial masses," which are long retained as centers of growth, and the presence of which makes regenerative changes possible (Fischel). These differentiation disturbances in the central nervous system have also been demonstrated in humans, and the question of the development of tumors from heterotopic tissue has been earnestly discussed (W. Vogt, and particularly Ranke, lit.). Not rarely, the first manifestation of a glioma is in early childhood, even in the newborn; most prefer the earlier years, but have been described in the sixth decade. Hoffmann saw gliomas in two sisters 33 and 48 years old respectively.

In some cases, a trauma has been accused of an illy defined influence, if not on its origin, at least on its growth (lit. by Flesch, Hunziker, Eppinger and others). In a very unusual case described by Merzbacher, a glioma arose 3½ years after a severe injury to the skull at the situation in which a sarcoma had developed; the irritation of the sarcoma is said to have initiated the glioma, but Landau, probably correctly, insists that there was a particular predisposition on the part of this brain similar to that of the skin which produces keloids instead of ordinary scars. Later, in a publication by v. Ujeda, Merzbacher leaves open the question of the actual glious new formation. A particular predisposition to glious scars is probably present in cases such as that observed by the author in a 19 year old man: a glioma as large as a hen's egg was found in the upper parietal part of the brain in the exact situation of a depression of the skull which had dated from a much earlier trauma. He was operated by C. Hagler, who later was present at the autopsy. This case—it was a poorly cellular, richly fibrous, glioma durum —is of considerable interest, because the severe trauma had left behind a very plain residuum. Even though Ribbert is correct in emphasizing that spongioblastic rosettes and spaces in a glioma point to a developmental disturbance, it does not necessarily follow that poorly cellular, richly fibrous gliomas are always a result of developmental disturbances, and cannot, as Ribbert says, be produced by trauma. In judging of the relationship between trauma and a glioma, it is probably necessary to consider the histological character of the glioma. See also Rossle, Nippe (lit.), Krauss, Durck, Frank, Huebschmann (lit.), concerning the question of traumatic gliogenous tumor formation; see also literature under sarcoma, p. 1933. Bartel and Weichselbaum call attention to the not uncommon finding of status lymphaticus and glioma as an expression of a hypoplastic constitution. The author can confirm this coincidence.

Ganglion neuromas (ganglionic neuromas or gangliоglioneuromas) have been but rarely observed in the brain; they commonly appear in the

peripheral nervous system, particularly in the sympathetics and in the adrenals (p. 1287). They are believed to originate from embryonal anlage cells in the central nervous system and have been observed in the temporal lobe: 2 cases of Schmincke, the first of which was called ganglioglioneuroma, the second, ganglioneuroblastoma, because immature cell forms were present and played a considerable part in the picture; this tumor thus combined the elements of neuroblastoma and pure mature ganglioneuroma (see p. 1288); and in the parietal and the frontal lobes: (2 cases of, Olivecrona), in the septum pellucidum: (Berblinger, large cells surrounded by glia fibers); in the floor of the third ventricle with involvement of the optic chiasm: (Robertson); and in the medulla oblongata: (Bielschowsky and Pick).

Tuberous sclerosis (p. 1908) must be differentiated from true tumors (gliomas) as a hypertrophic or hyperplastic form of gliosis (Marburg). Glioma and glioses (neuroglia hyperplasias) are differentiated—apart from the actual tumor characteristics such as sharp boundaries and progressiveness—by the greater abundance of cells and vessels in gliomas, which produces a relatively soft consistency, while in glioses the tissues are pale and hardened. As a rule, nerve fibers and ganglion cells are entirely absent in gliomas, and usually present in glioses. See p. 1842 for ependymal gliomas, and p. 1908 for ependymal gliomas in tuberous sclerosis.

See Uhthoff, and the critique of Brunner concerning the beneficial inhibiting influence of x-rays on blastomatous glia overgrowths.

Other Tumors

Sarcomas in general arise much more frequently from the meninges, the periosteum, and the bones of the skull than from the brain itself (fig. 918, p. 1838); when they do originate from cerebral substance, they produce round, soft nodules of variegated appearance, usually tougher and more sharply outlined than gliomas. The tumor often compresses the neighboring tissues and is separated from them by a so-called encephalitic softening zone. The tumor is easily enucleated.

Histologically, it is composed of spindle, or polymorphous cells, of larger or smaller, rounded, stumpy, spindle cells, etc. Since the cells are easily destroyed, teased preparations show only free nuclei. The cell masses of angiosarcomas form thick collars around the vessels; Minelli described a rare melanotic perithelioma, which is said to have arisen from the chromatophores which normally are present in the tissues around the vessel walls. Berblinger (lit.) believes that a melanotic tumor in the cerebrum, which occurred with multiple melanomas in the skin and other organs of a 9 months old child, arose from neuroepithelium which had the ability to form melanin a specific property possessed only by epithelium.

Sarcomas with the structure of endotheliomas have also been described; the cells are in layers around vessels or vessel buds. Telangiectatic forms, as well as myxosarcomas, have also been described. Glia masses may be retained in the edges of sarcomas and lead to the erroneous assumption of a gliomasarcomatodes or a gliosarcoma in the narrower sense. Fibrin networks in sarcomas may easily be mistaken for glia (Stroebe). The point of origin is in the blood vessel connective tissue apparatus. Etiologically, traumata are occasionally responsible; the author described a typical case of this kind, (Adler and others, also Bluhdorn, Reiche, and compare literature under glioma and trauma, p. 1931).

The differentiation of sarcoma and glioma is sometimes impossible macroscopically. Sarcomas of the cerebrum, however, soon invade the meninges (or arise in these), and are therefore frequently adherent to the soft meninges, and occasionally to the dura also, and to the bones which become infiltrated with fungoid masses. Poorly cellular gliomas prefer the white matter, and localize in the cerebrum allowing the soft meninges to remain intact. There is no essential difference between the rapidity of growth and wealth of cells in sarcomas and richly cellular gliomas; poorly cellular gliomas grow slowly. Microscopically, the demonstration of the syncytial structure (Stumpf) and of glia is sufficient to clinch the the diagnosis of glioma. Veszpremi recommends the use of Weigert's neuroglia stain, but in the experience of the author, iron hematoxylin (van Gieson) is sufficient. Gliomas infiltrate and use the already present meshes of glia in which to spread and are therefore not sharply outlined from the neighborhood; this is true also of the richly cellular, poorly fibrous gliomas, the so-called gliosarcomas (Stumpf), while sarcomas destroy the glia network, so that they are sharply outlined from the nervous system (but not always), and are separated from the surrounding normal tissue by a zone of softening. The tendency to hemorrhage, necrosis, softening, fatty degeneration and the formation of false cysts (p. 1936) is present equally in sarcomas and gliomas.

Metastases occur in the brain itself and in distant organs.

Wohlwill described a glioma of the cerebral hemisphere, and a sarcoma of the lenticular nucleus in the same brain.

Rare Primary Tumors in the Cerebral Substance.—Angiomas produce foci of red color, are composed of newly formed vessels which may be dilated and cavernous. They are often localized to the cortex, and their pial origin is unrecognizable; others are made up of multiple small foci (cavernous hemangiomas), chiefly in the white cerebral substance (Creite, Huebschmann). A congenital developmental anomaly is a possible source of their origin. They appear as circumscribed, often encapsulated, foci

of a meshwork filled with blood, usually of small, but occasionally extensive size, as big as a fist in a case of Lechner (lit.); see illustrations by Astwazaturoff. Calcification is visible on x-ray examination (Uyematsu). They also occur in the pia. Corten described a hemangioma sarcomatodes. A circoid angioma is very rare, *i.e.*, an angioma arteriale racemosum (Simmonds, Sternberg, Stertzing, Abrikosoff, Ranzel, lit., Therman, lit. and critique by Schmolck, Leunenschloss); a unique case was described by Simmonds as an "angioma arteriale serpentinum," in which the snaky structure developed along the course of an artery. In the cases of angioma racemosum described by Kalischer and Therman, the new formation involved chiefly veins and capillaries (see pp. 1840 and 151). Myxomas appear as large soft transparent tumors, the centers of which may soften and thus contain a mucinous fluid (pseudo cyst). Osteomas, slow growing and latent clinically, and fibromas, small, round and hard, are both rare. Carcinomas, which occasionally arise in the walls of the ventricle, have been mentioned on p. 1843. Huebschmann (lit.) described a peculiar malignant epithelial tumor of the temporal lobe as large as a hen's egg; it occurred in a 35 year old man in whom there were numerous metastatic nodules in the lungs and pleura.

Metastatic Tumors.—Carcinomas; especially the malignant chorion epitheliomas (see p. 1739 and fig. 952). The first manifestation of the latter may be an unlooked for apoplexy and hemiplegia (cases of Krewer), after the primary uterine tumor has been entirely removed; it is even more astounding if no tumor is present in the uterus (case of Busse in which the hemiplegia appeared 6 months after an abortion). The stroma in metastatic cerebral carcinomas is always scarce, and the nodules are relatively soft in the brain, because of the lack of connective tissue there; there is, however, an adventitial connective tissue present (Klippel and Renaud). Occasionally, the nodules are loosened by an encephalitis in the neighborhood. When they involve larger parts of the brain, brain pressure symptoms are quite frequent.

Sarcomas.—Among the sarcomas, those which arise in the skin and bones, and melanosarcomas, produce the most frequent secondary tumors. Malignant hypernephromas also (p. 1401) metastasize frequently enough in the brain. The size of the metastatic tumors varies between those scarcely visible to those as large as hen's eggs. As a rule, nodules are formed, but occasionally a diffuse spread is observed over the meninges of the brain and spinal cord.

Accurate statistics concerning the frequence, the number, the situation and the point of origin of metastatic brain tumors (cerebral substance and the meninges) are obtainable from the pathological institute of Basle. According to Krasting (lit.), in 1078 carcinomas with 817 removals of the

brain, only 39 cases had metastases in the brain (4.77%); in 160 sarcomas with 118 brain autopsies, 14 were found to have brain metastases or 11.86%. In ⅓ of the cases (Basle, and the lit.), the carcinoma metastases were multiple in the brain; the solitary tumors were more frequent in the left hemisphere; the cerebrum was more frequently attacked, the cortex more often than the white matter; the most striking finding was the preference for the left central convolutions. Among the sarcomas, most of the metastases were multiple without reference to any part of the brain; the relatively younger individuals were more frequently attacked, males more often than females. The carcinomas of the various organs which metastasize most frequently to the central nervous system are as follows (39 cases from Basle and 106 from the lit.): malignant chorion epithelioma, carcinomas of adrenals, pharynx, jaw, prostate, breast, bronchi and lungs, thyroid, sigmoid, gall bladder, rectum, uterus, stomach. For more

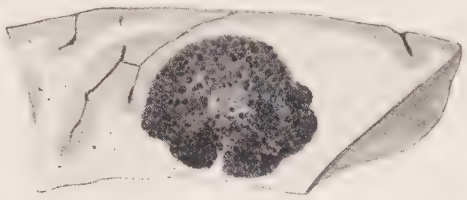


FIG. 952.—Metastatic chorionepithelioma in brain; from the case figs. 874 and 215; nat. size.

material concerning brain metastases, see Gallavardin-Varay, Lewin; special remarks concerning metastases from uterine carcinoma by Offergeld; female genital tumors, Schottlander (lit.); metastases of the malignant tumors of the central nervous system by Heinemann; carcinoma metastases in the cerebellum, Girardi.

In 23 cases of lung and bronchial carcinoma, the author, contrary to the prevailing opinion, found only one case with brain metastases (C. f. Schw. 17, 1906); among 6 cases discussed on p. 470, there was only one. From a colloid bronchial carcinoma, the author saw innumerable, cystic, cerebral metastases which showed the greatest similarity to old hemorrhagic, softened cysts; some were as large as pigeon's eggs, containing thick, clear, fatty, cloudy, or brown fluid. Only a few had an edge of finely nodular, richly vascular, tumor masses. Smaller metastases were solid. Occasionally the metastases are of microscopic size.

The relationship of secondary and primary brain tumors is brought out as follows: among 144 cases of brain tumors, 92 were primary (63.19%), 53 were metastatic (36.81%).

Cysts, (false and true), in the brain arise from various causes. (*a*) Cysts from spontaneous and traumatic hemorrhages, and traumatic

simple, and inflammatory softening foci (cysts following gun shot wounds, see Sultan). (*b*) They form by softening and hemorrhage in tumors (primary and secondary), among which may be mentioned gliomas, sarcomas, carcinomas, myxomas; (these false cysts, particularly frequent in gliomas and sarcomas, may reach the size of hen's eggs; they contain clear serous or fibrinous fluid). The inexperienced observer may not recognize their tumor nature, since the tumor has disappeared to a thin peripheral layer of the cyst. Microscopic examination is necessary. (*c*) Cysticerci and echinococci may give rise to cysts. (*d*) Diverticulum formation in the cavities of the brain (Virchow, Lichtheim; see also Auerbach-Grossmann, Henschen and others). (*e*) They may be formed by ependyma adhesions snaring off parts of the lateral ventricles, particularly the posterior horn. (*f*) Dermoid cysts are very rare (Ziehen, Raymond). (*g*) Gas cysts with gas-filled spaces in the brain, often in large numbers and measuring several centimeters in diameter, arise through the activities of gas-forming bacteria, and may be observed in that bacteremia in which gas emphysema is present in many organs. Many bacilli are found in the circumference of the spaces (Howard). See also K. Fraenkel concerning the various kinds of brain cysts; concerning cerebellar cysts, see Scholz (lit.), and Bartel and Landau, who correctly believe that most cysts are a result of regressive changes in gliomas; see also Krause, Tooth, O. Hildebrand. See also under porencephalia, p. 1860. For plexus cysts, see p. 1841.

The **tumors of the intracranial nerves** may be mentioned in this place. Neuromas (neurinomas, seen under nerves), neurogliofibromas, gliofibromas, fibromas, sarcomas, are particularly frequent along the acoustic nerve, and often thrust tags into the internal ear. (These have been illustrated by Knoblauch, Diss. inaug. De neuromate, etc., Francof. ad Moen. 1843.) Occasionally, they are bilateral. It is believed that they originate in tissue rests in the region of these nerves. They usually appear in individuals of middle age. The clinical picture is characteristic in these so-called cerebello-pontine-angle tumors, or tumors of the recessus acustico-cerebellaris, where cholesteatomas also occur (see fig. 919); see Oppenheim, Sternberg, Hartmann, Leischner, Bregman, and Krukowski, Henneberg-Koch, Henschen, Josefson, Pascalis, Berlstein and Nowicki, Henschen. Death following operations to remove these tumors is often due to shock and paralysis of vital centers.

Generalizations on the Consequences of Brain Tumors.—Many brain tumors when they have reached a certain size are accompanied by an internal hydrocephalus; but they also produce brain pressure symptoms without this condition. Gliomas which push aside the normal tissues in the beginning of their growth, may give no symptoms for a long time

(Redlich). The cerebral substance, as a rule, is firm and dry (Reichardt). The fluid and the venous blood supply are compressed. Signs of brain pressure are shown on the inner surface of the vault of the skull by ulcers and osteoporosis (the bone is red), or by thickening of the vitreous table; oval or rounded furrows are separated by steep, irregular, sclerotic combs. Fine, fibrous adhesions develop between the meninges and the dura (for calcium transport, see p. 1821). Ulcers may appear in a few weeks. Occasionally the cerebral substance is forced like a hernia through fissures in the dura which are widened by stasis in the enlarged Pacchionian granulations, and thus pushed into the Pacchionian furrows of the skull (Beneke), where it becomes soft and pulpy; these multiple cerebral hernias (von Recklinghausen) are particularly numerous in the anterior and middle fossae, often in parallel rows running from before backward. For more details concerning physiological brain hernias, see Wojno. For compression of the papilla-like parts of the cerebellum into the foramen magnum, see p. 1848.

The symptomatology of brain tumors is similar clinically to that caused by: primary and secondary tumors of the cerebral substance and the meninges; cysts of the plexus; tumors of the hypophysis; chordomas of the base of the skull (p. 1202); tumors of the bones, etc.; tubercles and gummata; cysticerci and echinococci.

General brain pressure symptoms are as follows: the most reliable are choked disk and optic neuritis (Uhthoff, Saenger, Reichardt), which, in general, develop earlier the more posterior the tumor, therefore are particularly frequent in tumors of the cerebellum or in internal hydrocephalus (Freeff), but also frequent in brain abscesses and meningitis; among the other symptoms are headaches, directly or indirectly always a meningeal sign (Marburg); numbness, sleepiness, and, less constantly, vomiting, dizziness, slowing of the pulse, general convulsions. Symptoms of brain pressure may be absent, even in the presence of quite large tumors, while, on the other hand, they may be present in small tumors if located in certain positions (Reichardt). A favorable change in the clinical picture may be caused by regressive metamorphoses and the clearing away of destroyed masses of tumor (Anton), or perhaps by the passage of the cerebrospinal fluid under pressure through foreign passageways (eyes, nose).

Focal symptoms; these, in part direct, in larger part indirect (p. 1889), are dependent upon the particular situation of the tumor and cannot be discussed in detail. It might be mentioned that tumors in the motor region can be diagnosed and localized earlier, and are occasionally successfully removed; they produce the most outspoken symptoms, which, as a rule, appear first as cortical epilepsy. Then paralysis is added, almost

always as a monoplegia, which, with the growth of the tumor, gradually becomes a hemiplegia (Oppenheim, Lehrb. 5. Aufl. 1908).

Comprehensive clinical discussions and literature concerning cerebral tumors by Allen-Starr, v. Bergmann, H. Duret, Bruns, Ballance, Biro, Redlich.

According to Weber and Schulz, the symptoms of a local space-compromising cerebral process can also be caused by "pseudotumor cerebri" (Nonne), among which may be mentioned damaged circulatory conditions of the brain from arteriosclerosis, perivascular infiltrates, hemorrhages and edema, destruction of pial cisterns, etc. Rosenthal saw fairly extensive amoeboid glial changes, but Reichardt doubts that this change, which takes several hours to develop, is present, as a rule, in swelling of the brain. According to Reichardt (lit.), a pseudotumor cerebri (Nonne) may be summed up in largest part under the conception of brain swelling. See also Nolen.

Meningitis serosa circumscripta cystica (p. 1825), as well as an acquired hydrocephalus (p. 1849), may simulate tumor; see Nonne concerning the former.

Parasites.—Cysticerci (p. 848) appear as vesicles, in which there is a cloudy point, the head and the long neck of the parasite; or in the form of calcified nodules in the brain substance surrounded by a connective tissue capsule, particularly often in older people and in the insane. Usually, they are multiple, and very often numerous. They prefer the cortex and the subcortical layers, particularly of the convexities. They reach the smaller arteries of the pia and are carried into the branches leading into the cortex. Only a few reach the larger arterial branches which supply the larger ganglia. According to Jacobsohn, they are present in the vessels, which produce the capsule of the cysticerci, but this idea is erroneous (Krause, lit., Margulis); the capsule is formed from granulations which become connective tissue. In general, the vesicles are small, no larger than the heads of pins or peas, but may reach the size of walnuts. In a patient who had innumerable vesicles in the subcutaneous tissues, in the meninges and in the brain, the author found a cyst in the white matter of the left hemisphere as large as a hen's egg. The slow growth of the vesicles makes it possible for the cerebral structure in the neighborhood to remain entirely indifferent to their presence; it is merely compressed and pushed aside; occasionally, however, softening, with death of the tissue, occurs in the neighborhood and this is followed by a reactive capsule formation. A sclerotic layer of glia may surround the capsule. The encapsulated cysticercus often undergoes fatty degeneration, calcification, and dies; rarely it suppurates. In a 24 year old man, the author saw numerous suppurating cysticerci in the cerebral substance,

appearing as gray red or yellow soft nodules as large as peas and resembling gummata or tubercles; microscopically there were fibrosis, fibroblasts in the vessels, plasma cells and lymphocytes infiltrating the capsule, about which there was a thickened zone of glia which was likewise infiltrated

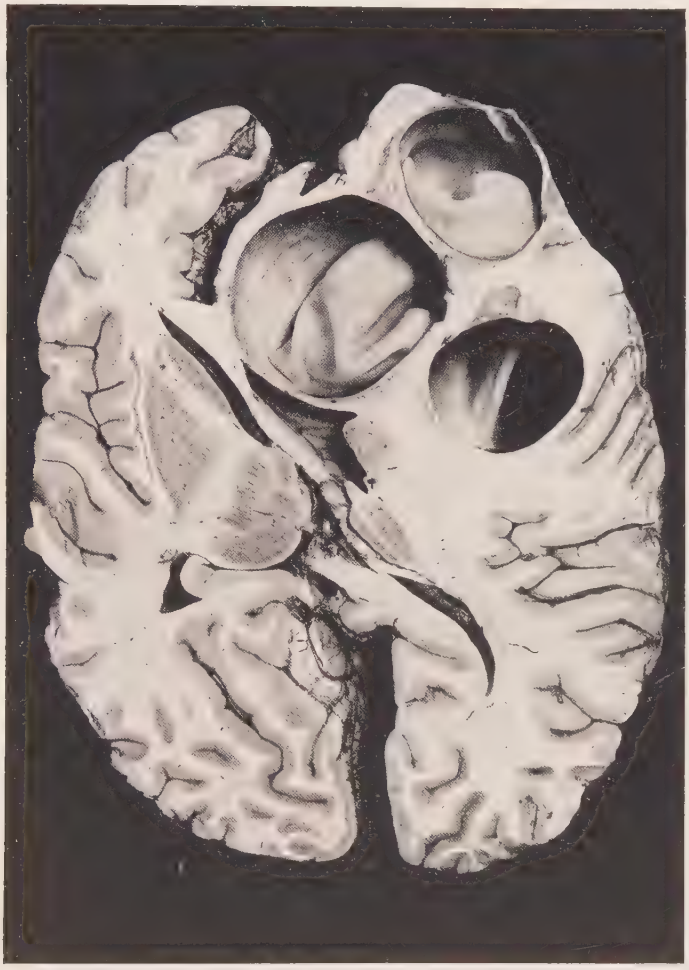


FIG. 953.—Echinococci in the l. cerebral hemisphere. Horizontal section showing the displacement of r. hemisphere and distortion of lateral ventricles, fornix and corp. callosum. From a painter 31 years old, ½ year before death psychic disturbances; 1 month before death symptoms of brain pressure (choked disk; lumbar puncture pressure, 220 mm.; coma, death). (Calcified echinococci in adhesions of pericardium, one the size of a walnut in wall of l. ventricle); ¾ nat. size.

by lymphocytic cells; the internal surface of the fibrous capsule was composed of granulation tissue with isolated phagocytes which were wandering toward the detritus of the parasite inside of the cyst; there were no giant cells, but a few isolated eosinophiles. In the clinical diagno-

sis, the finding of cysticerci in the skin is important (see under skin). Most frequently the vesicles are in the meninges along the convexities of the surface of the brain (p. 1845), occasionally free, or adherent in the ventricles (p. 1852).

Echinococcus hydatidosus (p. 971) appears as isolated, sharply outlined vesicles as large as fists, surrounded by a connective tissue capsule coming from the neighborhood; occasionally the vesicles are multiple. They are situated on the surface, in the cerebral substance, or in the ventricles; are particularly frequent in younger individuals and clinically may produce symptoms of brain tumor. In rare cases, there has been spontaneous rupture through the skull, the nose, the ears. An echinococcus cyst may be present coincidently in the liver. (Lit. by Steffen, Bruns, Schlagintweit, Gothard-Riche, Oppenheim.)

Echinococcus multilocularis (p. 973), usually situated in the frontal brain, has been observed in but few cases. The male sex is preferred, and those who take care of domestic animals (Melnikow, lit., Posselt, lit., v. Hibler). The foci in the cases reported were secondary and multiple, with the exception of the case of Roth-Bider which occurred in a 53 year old country butcher who suffered for six weeks with the symptoms of a brain tumor; the echinococcus was primary in the brain as a solitary spongy focus almost as large as a hen's egg, of irregular contour and separated from the neighborhood by connective tissue; it contained gelatinous material. Usually the primary lesion is a multilocular liver echinococcus, but in the cases of Hauser and von Hibler, they were present in the lungs (lit. by Melnikow, Posselt, Eichhorst). [For cystic blastomycosis of the cerebral gray matter caused by torula histolytica, Stoddard and Cutler, see Freeman, W., and Weidman, F. D., Arch. Neurol. & Psych. 9: 589: 1923.—*Translator.*]

Appendix. Observations Concerning Brain Pressure.— The arteries which lie in the lymph spaces filled with cerebrospinal fluid exert a pressure on this surrounding fluid each time they are filled, *i.e.*, during systole; these spaces on their part exert a pressure on the subarachnoid spaces and the veins in them, which, as a consequence, quickly empty into the sinus. Increased blood pressure produces increased pressure in the arteries and cerebrospinal fluid; the latter exerts an increased pressure on the cerebral substance, especially when the outlets for the cerebrospinal fluid (p. 1805) do not permit a sufficient amount of the fluid to make its exit, thus toward the subarachnoid space of the spinal cord. Conditions may occur in which the brain pressure is increased without this compensatory mechanism functioning; then the symptoms of brain pressure, among others, choked discs, become manifest. This, in turn, since there is a higher degree of tension in the fluid, exerts a higher pressure on the vessel

walls and thus hinders the arterial flow, whereby the blood supply becomes diminished (dysdiamorrhysis and adiamorrhysis, Kocher), and the nutrition and physiological functions of the brain are damaged. Increase of intracranial pressure is brought about by space reducing processes within the skull, or by stretching of the brain substance itself. Among the causes of the former are traumata (depressions), dural tumors, bone tumors, hematomata of the dura, etc. A quickly developing compression, as *e.g.*, from a rapidly growing tumor, frequently leads to anemia and softening of the compromised parts. Small, slowly growing dural tumors may compress the brain substance without producing severe degeneration, and symptoms of brain pressure are usually absent. Reduction of space, as occurs, *e.g.*, from hyperostosis of the skull, appears so slowly and is accompanied by such successive atrophy of the brain that brain pressure symptoms are often unnoted. Brain pressure is also caused by hemorrhages, tumors, abscesses, serous and suppurative meningitis (Hauptmann). See pp. 1850 and 1937 for the anatomical changes from brain pressure.

Traumatic Diseases of the Brain

Concussion of the brain may be caused by the action of blunt trauma on the skull as during a fall, push, etc.; according to Kocher, it consists of a suddenly exerted compression of the brain. It may lead to cerebral paralysis without gross lesions in the brain, (in some cases they are, of course, present), and produce transient unconsciousness and general motor paralysis, which may last for days or even result fatally, (Kocher).

For other conceptions of the mechanism of cerebral concussion, see Rahm and, opposed, Breslauer-Schuck.

In severe concussion of the brain all psychic functions are abolished. The pulse is slow, small in volume; breathing may be normal, or superficial and irregular. The skin of the face and extremities is pale, the bodily warmth is diminished. The pupils are dilated, react poorly or not at all to light. Transient vomiting is an almost constant symptom. (For further clinical notes, see Trendelenburg, Windscheid, Demmer and others.)

The cause of death in concussion of the brain when the anatomic findings are negative is supposed to be shock, (see p. 1888); others believe that during the concussion, all the parts, especially the centers, are injured mechanically in the same way as the vaso motor center (through irritation and paralysis) (Koch and Filehne). But that gross damage is done, such as multiple hemorrhages (tearing of vessels through shock or the push that the brain substance resists), is shown by the fact that foci of white softening appear as an expression of the microscopic damage to the nervous

tissue (Schmaus, Jakob), to which may be added petechial hemorrhages, or those of larger degree (see traumatic late apoplexy, p. 1943). According to Ricker, the latter occur by diapedesis following changed irritability of the nervous system. Occasionally, calcified ganglion cells (p. 1871), focal atrophy, scars or cysts, point to a previous local concussion or contusion without external injury to the head. It may also be mentioned that, according to Schmaus, in analogous fashion, concussion of the spinal cord may lead to death of nerve fibers, and that traumatic necroses also lead to softening and later hemorrhage in this structure.

Molecular changes in the central nervous system which cannot be demonstrated anatomically are said to be the cause of the functional disturbances which are grouped under the term "traumatic neuroses." According to Oppenheim, these occur from psychic and physical concussions which involve particularly the cerebrum. They have often been observed after injuries in trains ("railway spine" or "railway brain"), but may, of course, occur in other accidents. In a few cases, changes in the smaller vessels have been demonstrated throughout the entire brain; they consisted of aneurysmal dilatations, hyalin changes, round cell and pigment infiltration in the lymph sheaths, etc., (Sperling, Friedmann and others). The opinions concerning these traumatic neuroses are much divided (see Schuster). But that contusions to the back in railway accidents may produce changes in the spinal column and the spinal cord has been shown beyond doubt by more delicate investigative methods (see, for example, B. Payr). After direct trauma to the head there always occur finer changes in the cerebrum; the most constant are said to be round cell infiltration of the vessel walls (Yoshikawa). For the experimental aspect, see Jakob.

Shattering of the brain may occur from violent local concussion or from compression (contusion), in fractures, after blows or falls on the skull—concussion and contusion are very frequently combined; the picture is usually that of red softening of cortical parts which are pulpy and infiltrated by punctiform hemorrhages.

Microscopically, the capillary bleedings (diapedetic) are ring-shaped hemorrhages as described on pp. 1836, 1883 (Kirschbaum).

The traumatic red softening is reabsorbed in time, if it remain sterile. This may take a very long time, but it does occur finally, just as in spontaneous softening and focal hemorrhages, perhaps leaving behind a pseudo cyst (p. 1890). But it must be emphasized that degenerative changes from trauma often spread far beyond the actual situation of the wound. This is at the basis of the traumatic late apoplexies; according to Bollinger, progressive softening necrosis follows traumata; these produce alterations in the vessels, which later lead to hemorrhages,

analogous to those which occur in the spinal cord (Schmaus, see opposed, Ricker). The time between trauma and apoplexy varies from weeks to months. Langerhans assumes, probably erroneously, that aneurysms have developed in such places (lit. by Schuster and Densow). If a foreign body is present as, *e.g.*, an embedded splinter of bone, even though it is bland, the focus may become progressive and lead to extensive cerebral softening.

Blunt trauma to the skull, as from a blow or a fall on the head, may crush the brain directly or indirectly (contusio cerebri). If the blow is severe as, *e.g.*, from a crowbar, both the skull and the brain may be injured at the point of contact of the weapon. But if, for example, an individual falls with considerable force and strikes the posterior part of the head, the skull is fractured at the point of contact, but also parts of the base of the skull with it, while the brain with its soft meninges is not necessarily injured at the point at which the blow was struck, but perhaps at a situation where it meets with resistance as the force is carried forward, *i.e.*, after a fall on the posterior part of the head, the brain is injured at the base of the frontal cerebrum and the anterior part of the temporal lobes; in these situations the brain is superficially splintered (red softening); this is called contrecoup. Residues of such contrecoup in the form of plaques jaunes, are seen most frequently in the parts of the brain which rest upon the roof of the orbit and the middle fossa. An indirect or a contrecoup fracture of the bone is not frequent. More rarely, deeper parts such as the walls of the third and fourth ventricles, the aqueduct, the septum pellucidum are ruptured by severe concussions. In severe injuries to the head, such as in railroad accidents, blows on the head with blunt instruments, falls on the posterior part of the head and fractures of the base of the skull, hemorrhages may occur in deeper parts of the brain. They are usually of limited extent, but the author found a hemorrhagic focus as large as a hen's egg in the right frontal region of a drunkard; occasionally they are multiple, sometimes in rosary form, as small rounded petechiae. In their differentiation from spontaneous apoplexy, the situation must be taken into account, since spontaneous apoplexy, as a rule, occurs in the depths, in the neighborhood of the corpus striatum. Furthermore, there is practically always an intrameningeal hemorrhage in contusions; very frequently small splinters of the skull can be found, at least in the more severe traumata to older persons. Spontaneous hemorrhages are usually more extensive, and seldom appear without other changes, such as shrunken kidneys, hypertrophy of the heart, arteriosclerosis, etc. Hemorrhages into the brain after injuries to the head occur, of course, more readily if the vessels are arteriosclerotic.

In fractures of the base of the skull, hemorrhages from the ears are not uncommon. The blood comes most frequently from the jugular vein or

the transverse sinus, which lie close to the wall of the tympanic cavity; the blood runs through the torn ear drum into the external ear. Traumatic hemorrhage from the ear may also occur, however, with the drum intact, as the author has seen repeatedly at autopsy, when the fracture is sagittal through the mastoid process; the external auditory canal is torn at the transition place from the cartilaginous to the bony part.

Cerebral wounds from stabs or gun shots and projectiles, if they remain aseptic, heal both by granulation and scar formation, and by growth of the glia. This process continues until all the destroyed parts are resorbed and their situation cicatrized, even though it takes years (details by Blum). Larger defects leave cysts (p. 1890).

Consult the experiments of Tanaka.

Perforating gunshot wounds which are filled with a pulpy mass of destroyed cerebral substance and surrounded by a bloody wall, may later be lined by granulation tissue, and finally become fibrous. If the trauma opens the skull, the local differences in pressure force the cerebrum outward (primary, pure, physical prolapse). In the later course of events, there is acute inflammatory swelling of the brain (encephalitis), and jutting through the opening of the skull are soft hyperemic, or hemorrhagic, cellular infiltrated "polypi," as secondary brain prolapses, or "fungus cerebri" (Schrottenbach, lit., Borst).

The cases of prolapse following infectious, progressive encephalitis, (malignant prolapse, Payr), are almost always fatal because of rupture into a ventricle (p. 1907) or the development of a fistula into the ventricle into which the brain prolapses.

A bullet, particularly from a revolver or other weapon with relatively less force, often produces not a simple, but an angular canal, because it passes through the brain, strikes the wall of the skull opposite to its point of entrance, tears the dura, leaves an impression on the bone (or if death occurs late, a small osteophyte), then richochets at an angle to again reenter the brain for a short distance. Occasionally, it is deflected back into the original path. The path of the bullet through the brain is usually spindle shaped; the entrance is larger than the exit. The gray matter which is more vascular and less dense than the white, is subject to more destruction than the white matter (Tilmann).

Infection of cerebral wounds, as a rule, leads to suppurative infiltration, abscess formation (p. 1903), internal pyocephalus following rupture of an abscess, acute brain edema with sudden permeability of the abscess membrane, or meningitis over the abscess, or over areas of a pyocephalus. The infection may occur spontaneously, as *e.g.*, from the nose.

See Ricker and Ghon, Hart, lit., Benda, for war injuries to the brain.

Hypophysis Cerebri and Glandula Pinealis

For the development, see pp. 1953 and 1954.

The **hypophysis or pituitary gland** is a structure of variable size, lying in the sella turcica, consisting of two lobes. The chief mass of this endocrine body is the larger, anterior lobe, composed of the epithelial gland-like part, built up of numerous rounded and elongated alveoli or follicles, some of which are filled entirely with epithelium, while others in the posterior portion are merely lined by epithelium and filled with a colloid secretion. These alveoli or follicles are surrounded by connective tissue septa and capillaries. It originates from the ectoderm of the oral fossa. Squamous epithelium, as ectodermal, but not epidermal, remnants of the hypophyseal ducts, may be included within the hypophysis (Erdheim). The posterior lobe is the nervous part (neurohypophysis); it is the direct prolongation of the infundibulum, arises from the central nervous system, contains a finely fibrillar interstitial substance, ganglion cell-like, and glious structures (Tello), nerve fibers, and sparse glia (Haberfeld) containing pigment (A. Kohn, Jonnesco, lit., Stumpf, Vogel). (See Tello for the more detailed histology of the neurohypophysis; for its various types, see Jonnesco.) The function of the posterior lobe is debated (lit. by Jonnesco); it is thought that the active secretion of the hypophysis is manufactured in the anterior lobe, is activated in the posterior lobe, and is finally excreted through the infundibulum (Gottlieb, lit.). According to Biedl, the secretion of the anterior lobe is sent directly into the blood stream (blood gland).

The commercial pituitary extract (pituglandol) is said to come from the anterior lobe, pituitrin from the posterior lobe, and coluitrin from the pars intermedia. The extract from the anterior lobe is growth stimulating, but according to Biedl, manifests itself only during the second period of puberty, in which 20 centimeters may be added to the height in one year, at the same time that development takes place of the secondary sexual characteristics. See, among others, M. Oehme, concerning the use of the extract of the posterior lobe in gynecology, as a styptic in hemorrhages, or in bladder weaknesses (post operative and puerperal), cardiac weakness, osteomalacia, see Bab; see Oehme and others, and p. 1949 concerning inhibition of diuresis by the extract of the posterior lobe.

The pars intermedia in the adult is very small in contrast to that of the fetus; it is composed of poorly vascular tissue containing islands or groups of cells frequently arranged around cavities lined by cubical epithelium, containing colloid, and resembling the alveoli of the thyroid. In addition, there are other spaces lined by ciliated epithelium (Weichselbaum), which represent remnants of the primitive hypophyseal space

(p. 1954); these may become cystic and contain colloid; colloid vesicles and glandular tubes may also invade from the pars intermedia into the neurohypophysis (Tolken, lit., Schaffer; Nothdurft has described a small basophilic adenoma of the neurohypophysis). See E. J. Kraus for staining methods to differentiate the colloid. According to Erdheim and Stumme, the anterior lobe contains three kinds of cells: (1) The chief cells (chromophobe cells) show large, rounded, or irregular nuclei, dark or somewhat lighter, with a weakly staining protoplasm that is hardly visible. They are quite similar to lymphoid cells. There are no cell boundaries. (These cells have been called chief cells by Stieda, nuclear masses by Rogowitsch, nuclear-rich protoplasm by Schoenemann.) (2) The eosinophilic (acidophilic) epithelial cells mostly contain small, always rounded, nuclei and sharply outlined sparse or rich protoplasm, which is thickly filled with granules intensively staining with eosin. (For more details of elective staining, see E. J. Kraus.) (3) The basophilic (cyanophilic) epithelial cells, larger than (2) have bright, rounded nuclei, (rich protoplasm), sharply outlined, with granules staining violet with hemotoxylin; (2) and (3) are also called chromophile cells (Flesch), because their protoplasm stains so intensively, as contrasted to the chromophobic chief cell. The relative number of these cells is as follows: the chief cells are the least numerous; they occur in all parts, occasionally in the center of an alveolus or a tubule, or isolated and surrounded by numbers of (2) or (3); the eosinophilic cells are the most numerous, especially in the posterior part of the anterior lobe; the basophilic are more numerous than the chief cells and lie mainly in the anterior part; they may even produce independent structures. According to Benda and Morandi, there are transitions between baso- and eosinophilic cells according to the different stages of secretion, but Erdheim does not confirm this view. According to E. J. Kraus, the eosinophilic cells are normally formed directly from the chief cells; under pathological conditions, (tumors), they are said to develop not only from the chief cells, but also from non-granular predecessors; the basophilic cells are said to arise from the chief cells either directly or through transitional cells, which are an intermediate form between the chief cells and the chromophilic cells; after elimination of their nuclei, the basophiles are said to be able to return to chief cells. Erdheim demonstrated fat granules in the cells; they increase with old age, both in number and size (Castelli). In middle life, the chromophilic granules are in the majority; in senility, the chief cells are predominant. According to Lowenstein (lit.), however, the chief cells are much more numerous in younger individuals than in older. According to Erdheim and Stumme, the weight of the hypophysis in the male and nulliparous woman varies from .56 to .64 grams; it reaches its maximum in the fortieth

year. (For more details, see Petersilie.) For the preservation of the cell structures by the injection of 10% formaldehyde solution through the optic foramen of the cadaver, (see Thomas), a strong needle is passed under the caruncula lacrimalis about 3 centimeters deep through the conjunctival sac.

Rogowitsch and Stieda, Hofmeister, Gley and others, in rabbits, and Izumi in cats, have demonstrated an enlargement of the hypophysis following total extirpation of the thyroid gland, which they believe to be compensatory; it involved the anterior lobe and only the chief cells, never the chromophilic structures. Others have demonstrated an enlargement of the hypophysis in humans following atrophy or aplasia of the thyroid gland in myxedema, but in only a few of the cases (Bourneville and Bricon, Osler, Calderara, lit., W. H. Schultze, Zuckermann, Berblinger and others). De Coulon found atrophy of the hypophysis in cretins; others found, contrariwise, an hypertrophy (p. 528); Eichhorst, at first, saw hyperplasia in the anterior lobe with a subsequent inflammatory change, hemorrhages and connective tissue overgrowth which led to destruction of the latter and pressure atrophy of the glandular structure. While Schoenemann in a large number of observations of degenerative processes in the thyroid gland saw similar degenerations in the hypophysis as parallel findings, Comte, on the contrary, saw hypertrophic and hyperplastic changes in the hypophysis in all cases of degeneration of the thyroid, a result which agrees better with experimental findings. The relation between the sex glands and the hypophysis is also quite interesting. Comte, among others, found an hypertrophy of the hypophysis during pregnancy, which Fichera believes due to a reduction in the function of the ovary. Then Erdheim and Stumme demonstrated that the previous gray red section surface became dull, gray white and moist during pregnancy, and that the chief cells were increased, and fat-free, granular, so-called pregnancy cells, appear, which, according to E. J. Kraus, are a particular variation of the eosinophilic cells. From the ninth month onward, they compose about 80% of all the cells of the hypophysis; the eosinophiles predominate only in the posterior part of the anterior lobe. As a rare case, Jung observed that a hypophyseal tumor disappeared after the interruption of pregnancy at seven months. In multipara, increase in weight up to three times the normal has occurred. In nullipara, from 21 to 40 years of age, the weight is about 61.8 cg; (for more details and literature concerning the weight, see Erdheim and Stumme, and also Kolde). See Berblinger concerning experiments done with material obtained from the membranes, placenta, and the fetus, in attempts to produce changes similar to those occurring in pregnancy. Marek believes that certain changes in growth such as plumpness of the face, the lips

and other parts of the body during pregnancy are a sort of physiological acromegaly. During the puerperium, the hypophysis becomes smaller, the pregnancy cells return to chief cells. Schmincke saw "pregnancy hypertrophy" of the hypophysis in secondary ovarian carcinoma; Karlefors saw similar, even though inconstant, changes in the hypophysis, but without hypertrophy, in all cases of carcinoma, (effect of the toxin of carcinoma?). Fichera found hypertrophy after castration in animals (capons, oxen); in steers, Schonberg and Sakaguchi (compare Wittek) found cell groups, even though inconstantly, composed of eosinophilic epithelial cells with very dark pycnotic nuclei. Tandler and Gross, (see also W. Koch), found an enlargement of the hypophysis radiographically in living humans who had been castrated as a part of the religious observances of the Skopzes in Russia and Roumania. Kon also, found an increase in the volume and the weight, and an increase in the eosinophilic cells in castrated individuals. The increase in the longitudinal growth of castrates (p. 1186), is believed by Tandler and Gross to be due to the hypophysis, and Exner (lit.), after transplantation of this gland, found increased secretion, increased growth in length and increased adiposity. Stumme (lit.) found the gland enlarged after destruction of the testes by pathological processes. In females after castration, even though inconstantly, Rossle saw increase, enlargement and heterotopia of the eosinophilic epithelial cells, with paucity or lack of basophiles. After extirpation of the hypophysis in young animals, Aschner found marked increase in the development of the genital organs; in adult animals he found sterility.

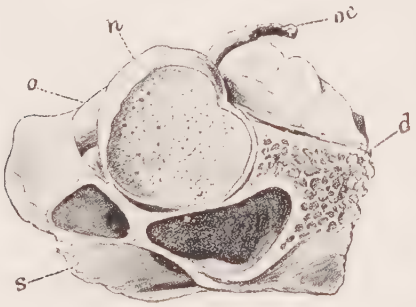


FIG. 954.—Adenoma (so-called struma) of the hypophysis cerebri (h), encroaching on sphenoid sinus, optic (o) and oculo motor (oc) nerves. Median sagittal sect. Below the struma, the sphenoid sinus, anteriorly the ethmoid (s); d, dura mater; from a 45 year old woman with carcinoma of parotid; nat. size.

By far the most important changes in the hypophysis consist of hyperplastic growths in the anterior lobe which lead to marked enlargement; these have been called adenomas or strumas of the hypophysis (fig. 954).

See Haberfeld (lit.), concerning ependyma epithelial cysts in the neurohypophysis of human fetuses, which are believed to be embryonal inclusions, and may possibly be the starting point of some tumors of the posterior lobe.

All other true tumors of the hypophysis are rare; carcinomas (Cagnetto, adenocarcinoma) and sarcomas are relatively the most fre-

quent; lipomas and cystic teratomas of the region of the hypophysis have been described (Beck, Gautier, lit.); the latter may easily be mistaken for tumors of the hypophyseal duct, (see p. 1952). The tumors of the hypophysis may reach the size of hen's eggs or larger. They exert pressure on their surroundings, particularly on the optic chiasm, which is displaced upward and forward. Damage to the optic nerves (see Zander) produces bitemporal hemianopsia, and even complete amaurosis (blindness); the oculomotor and the trochlear nerves may also be compressed and produce corresponding symptoms. But some of the tumors give no eye symptoms (Meggendorfer). These tumors may also invade the brain, rupture into the third, more rarely, into the lateral ventricles, or bore deeply into the temporal or other parts of the brain. The extra-sellar part may be far larger than the intra-sellar part (lit. by Roth). The bones may also be invaded (Erdheim, Verse), but metastases have seldom been observed, and then usually as regional lymphatic nodules, more rarely of wider distribution than into the neck (as in a tumor of the neck in cases of Fahr, Budde, lit.).

Tumor metastases in the hypophysis are not exactly rare, especially not in carcinoma. Schmorl saw most of them in the posterior lobe, Simmonds only in the anterior lobe.

The **diabetes insipidus** often observed (sometimes suddenly) in both primary and metastatic hypophyseal tumors consists of polyuria without sugar and with low specific gravity of the urine, caused, according to Leschke, by mechanical injury to the subthalamic region of the pars intermedia, the center of visceral innervation. Others believe that diabetes insipidus is an expression of a dysfunction of the entire gland, (consult, among others, v. Meyenburg); others believe it is due to disease of the posterior lobe (Eisner, Brandis), or of the pars intermedia (Schafer, Simmonds, lit.); and still others, that the disease is not merely a disappearance of the inhibitory function of the pars intermedia, but that it is necessary to have an intact anterior lobe, the secretion of which acts upon the kidneys, probably indirectly, through nervous influences (v. Hann, lit., Jacoby, lit.). Since Bab saw a prompt diminution in the amount of urine from fourteen to two liters per day in a case of diabetes insipidus, after he had given injections of hypophyseal extract (pituitrin), he believes that there is a hypofunction of the posterior lobe. See p. 1955 for hypophyseal cachexia from disappearance of the hypophysis in secondary and primary tumors.

In **acromegaly,** there have been found almost constantly (for exceptions, see Petren, lit. and the critique of B. Fischer), tumors of the hypophysis, usually described as adenomas, or tumor-like enlargements, or, at least, histological changes which indicate hyperfunction (see figs. 550 and 551).

The relations of the hypophysis differ in the cases of obesity and eventual genital hypoplasia, or the so-called hypophyseal adiposis or dystrophia adiposo-genitalis. These cases are rarely accompanied by acromegaly (Strumpell). (Lit. Frohlich, Weiss, Marburg, Strada, Gottlieb, see also p. 1951.)

The different kinds of adenomas of the hypophysis are: the eosinophilic variety, the most common of the anterior lobe (Benda), is the only one typical of acromegaly. The basophilic, as well as the chief cell adenomas, are rarer (Erdheim, E. J. Kraus, Benda, Lowenstein, lit.); their cells occasionally take on the character of pregnancy cells, (see Poindecker). E. J. Kraus has described transitional cell adenomas, and fetal adenomas, or adeno-like hyperplasias with immature cells. But acromegaly is brought about only by the eosinophilic adenoma, and not by the others, although the latter, according to Berblinger, may give rise to genital disturbances without adiposity. In adults, Priesel described tissue malformations coming from the primitive pharynx and extending into the neurohypophysis and the infundibulum, even up to the tuber cinereum; they are composed in part of polygonal epithelioid cells with indistinct boundaries, and in part of spindle-shaped cells with short out-runners, held together by single or multiple bands, or fasciculi, or networks containing many blood vessels and reticulum cells which may be stained by the newer method of Maresch. Small tumors arise from these structures; in the case described by C. Sternberg, it was believed that the cells were poorly developed neurogenous elements.

The sella turcica may be deeply scooped out (more details, by Erdheim); in the acromegaly illustrated fig. 550, the saddle pommels were as thin as paper. But the frontal sinuses, the maxillary sinuses, and the other air-containing spaces in this, as well as in other cases, were enormously distended because of atrophy. In fact, it has even been stated that the dilatation of the sella is primary, and the enlargement of the hypophysis is a growth ex vacuo (Jendrassik, Mitchell and Le Count), particularly because careful examination of the cases often shows a dilatation of the sella when only edema or a hemorrhage is present to fill it. In the cases of Waldow and others, there was said to be typical acromegaly with dilatation of the frontal sinus and of the antrum of Highmore; the hypophysis was practically normal. These cases, however, have not withstood criticism. Acromegaly without typical change in the hypophysis does not occur, and a differentiation into "pituitary" and "non-pituitary" acromegaly cannot be countenanced (Arnold, lit.).

The theory of a hyperpituitarism (Tamburini and others) as the foundation of acromegaly is now the best supported theory, and the genital atrophy in acromegaly is also a consequence of this condition (Exner).

The older views, which stated that there was an alteration in the secretory glands of the hypophysis as a cause for its growth (Stumme), whereby the secretions of the overgrown hypophysis, or, as Mayer says, "the changed conditions," bring about an acromegaly, have all been put aside. The eosinophilic adenoma of the anterior lobe of the hypophysis is now recognized as the factor leading to this condition; it produces a diseased, or an increased, or a changed secretion (hyperpituitarism or dyspituitarism). In relation to cases of the first kind, B. Fischer calls attention to an observation made by Lewis, in which the specific cells of the hypophysis were increased without general enlargement of the hypophysis itself. It is not surprising that there occur hypophyseal tumors without acromegaly (Frohlich, Carbone and Cagnetto, Kollarits, lit., Krumbhaar, Creutzfeldt, lit., Harbitz, Moskalew, Martius, Kahlmeter). The signs of acromegaly are wanting when the hypophyseal tumor is constructed of other tissues of basophilic and other non-eosinophilic cells, of endotheliomas, sarcomas (see, *e.g.*, Caussade and Laubry)—or when the tumor is in the posterior lobe, or in the hypophyseal duct, and furthermore, when the gland is destroyed by gummas or tuberculosis, so that true hypophyseal cells are no longer present. The successful result of operative removal of the hypophyseal tumor, first done by Hochenegg, is very important evidence for this theory, since the hands and feet become smaller, menstruation is re-established, and the thyroid gland enlarges. According to this, the relationship of acromegaly to the hypophysis is shown relatively simply. But there are said to have occurred cases of acromegaly in which there was no eosinophilic adenoma; moreover, small eosinophilic adenomas have been found quite frequently without the presence of acromegaly (Lowenstein, Erdheim and Stumme). B. Fischer believes these adenomas are too small to bring about this condition through hypersecretion; when they are larger, he believes there might be differences in the kind of secretion which cannot be demonstrated morphologically. E. J. Kraus emphasizes that it is necessary to have not only mere cell increase, but also a certain quality to the hyperfunction of the cells; the histological examinations are not always above criticism, and finally, many cases have been erroneously termed acromegaly, as for instance, the case of Petren.

Acromegaly is occasionally combined with splanchnomegaly (see, among others, E. J. Kraus, Amsler, Reinhardt-Creutzfeldt). Vice versa, hypophyseal cachexia, which is believed to be due to a-pituitarism, (p. 1955), is often combined with splanchnomicria, diminution in size of a number of the internal organs. For the combination of acromegaly with syringomyelia, see p. 1977, and Petren. (See the critique of B. Fischer, and the answer of Petren concerning this case in which the hypophysis

was not enlarged.) According to Falta, acromegaly is occasionally accompanied by marked growth of hair. Glycosuria is not rare in acromegaly; according to v. Noorden, it shows large variations. As an explanation of this diabetes mellitus, there is thought to be a hyperfunction of the anterior lobe, and an irritation of the hypophyseal stalk; or an interruption in the connection between the hypophysis and the brain, caused by tumors or trauma (Verron). For more details, see Aschner, who observed a severe glycosuria after injury to the floor of the third ventricle such as occurs only after the Claude-Bernard puncture at the base of the fourth ventricle. For acromegaly in lues, see Nonne, lit.

The relationship of hypophyseal tumors to adiposity (lit. Strada) and dystrophia adiposo-genitalis is still much debated. Erdheim (lit.) is of the opinion that the occurrence of the adiposity is not necessarily dependent upon the tumor in the hypophysis itself, but, *e.g.*, may come from the hypophyseal stalk, (see below), extend to the base of the brain, and through destruction or pressure in the neighborhood of the infundibulum, (or, as others believe, at the base of the third ventricle, or in the midbrain), damage the "vegetative centers" (see Aschner). The hypophysis occasionally undergoes complete atrophy from pressure of a tumor of the hypophyseal duct (v. Miller and others), but the resulting elimination of the hypophyseal function (hypofunction), is said to be of no particular importance, although Rossle described a case of dystrophia adiposo-genitalis in atrophy of the hypophysis without a tumor. On the other hand, B. Fischer (lit.) believes that damage (compression) to the infundibulum and posterior lobe obstructs the secretory stream from the anterior lobe into the stalk and into the brain (third ventricle); see Aschner, Stumpf, E. J. Kraus. Following partial extirpation of the anterior lobe in adult animals, adiposity and general atrophy have been noted (Cushing), while Goetsch-Cushing-Jacobson saw adiposity appear after removal of the posterior lobe. According to Biedl, obstruction to the outflow of the intermediate lobe toward the midbrain is the important factor in the origin of dystrophia adiposo-genitalis. At the same time, there is quite frequent enlargement of the thyroid gland (more rarely, a fibrous atrophy); this has also been seen in cases in which the hypophyseal tumor has been removed. Literature in appendix.

Hypophyseal duct tumors usually appear in the region of the infundibulum; they may be cystic, cystic papillary, or partly cystic and partly solid, benign or carcinomatous, sometimes resembling basal cell carcinoma, and usually, but not without exceptions, (see the cases of Bartels, Strada, Lindt), characterized by the absence of keratohyalin and cornification, which makes them similar to adamantinomas (p. 589) in this and in other respects, such as the hydropic changes in the epithelium (consult Sieg-

mund). They are often extensively calcified, have necrotic cells as in calcified epitheliomas (fig. 1058), with metaplastic bone formation in the connective tissue, hydropic swelling, and liquefaction and pseudo-cysts in the stroma. They were first described by Erdheim in his comprehensive work. They are never related to acromegaly nor to giantism, but may be coincident with adiposity and hypoplasia of the genitals (dystrophia adiposo-genitalis). Understanding of these relationships may be furthered by a brief review of the development of the hypophysis: the

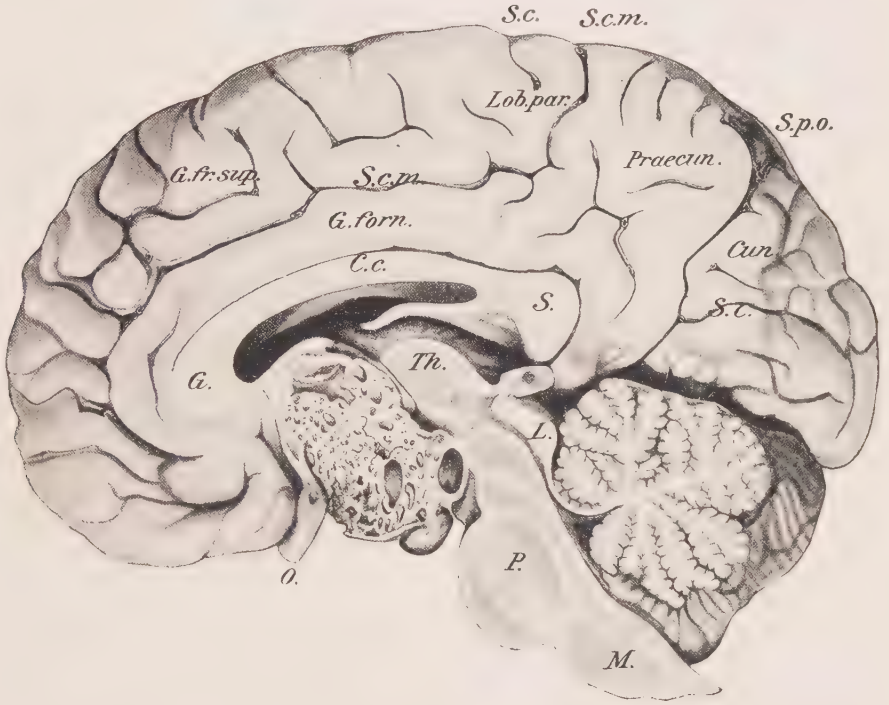


FIG. 955.—Small cystic epithelial tumor of duct of hypophysis from woman 51 years old. Approximately median sagittal section, some part of the convolutions of median surface of right hemisphere cut flat. G. forn., gyrus fornaticus; Praecun. precuneus and part of Cun, cuneus; Lob. par. paracentral lobe; G. fr. sup, superior frontal gyrus; Cc, corp. call.; G, genu; S, its splenium; below, the right lateral ventricle; the corp. call. and fornix are joined; Th, r. optic thalamus; L, lamina quadrigemina continuing backward into the ant. medullary velum; anterior to this the aqueduct of Sylvius, leading from III to IV ventricle; above it the epiphysis with small cyst inside; P, pons; M, medulla oblongata; O, opticus; Sc, central culcus; S. cm, calloso-marginal sulcus; Spo, parieto-occipital sulcus; S.c, calcarine sulcus; ¾ nat. size.

hypophysis is formed through a union of a basal part of the brain, the processus infundibuli, which gives rise to the posterior part, the neurohypophysis, with a protrusion into the base of the cranium coming from the primitive pharyngeal pouch which is lined by layers of cylindrical epithelium. As the bony parts of the base of the skull develop further, the open pocket soon becomes a vesicle, the hypophyseal sac, from which the anterior lobe of the hypophysis is formed through budding and snaring

off of solid epithelial masses. The early hollow hypophyseal duct soon becomes a solid strand of cells; the canalis craniopharyngeus (lit. by Sokolow, Gautier, and others), which passes obliquely through the sphenoid bone from behind and empties into the sella turcica, brings about this union. Erdheim could show that squamous epithelial cell masses are present in the hypophysis of 80% of adults, particularly in the anterior surface of the anterior lobe, but more in the prolongation, which extends parallel to the infundibulum almost to the chiasm. This represents the situation in the embryo where the ectodermal hypophyseal duct inserts into the anterior lobe of the hypophysis, and these cell masses are remnants of the duct which usually disappears during embryogenesis. Haberfeld (lit.) then showed that such "remnants of germinal cells" could be found along the entire path over which the hypophysis passes during its rise, even into extreme old age, as a macroscopically visible, strand-like organ under the mucosa of the roof of the pharynx, *i.e.*, as an organ composed of hypophyseal tissue which might be called the hypophysis of the pharyngeal roof, (see also Kilian, Civalleri, Pende). It also occurs as small rests between the roof of the pharynx and the base of the skull, or at the upper opening of the canalis craniopharyngeus (lit. by Haberfeld), or in the sella itself between its anterior wall and the hypophysis. Christeller (lit.) called attention to individual differences in the size, form, position, and particularly the histological make-up of the pharyngeal hypophysis, and raises the question of its compensatory ability in diseases of the main body of the hypophysis itself. That tumors may arise from such hypophyseal centers was shown conclusively by Erdheim (Bartels, Formanek, Masera, Ehlers, Strada, lit., see also Steinhaus). Erdheim differentiates benign tumors in the infundibulum mostly lying behind the chiasm, connected above with the base of the brain and below with the hypophysis, and malignant tumors (benign and malignant) developing in the hypophysis itself (Leegaard, Lubarsch, Teutschlander, Guizzetti, Nagoya, Lindt, Kankeleit, v. Miller).

According to Biedl, hyperplasia of the anterior lobe before completion of physiological growth leads to gigantism, afterwards to acromegaly. For more details, see pages 1186 and 1187, with literature.

See pages 1179 and 1180 for literature and remarks concerning hypophyseal dwarfism which is said to be caused by early diminution or elimination of hypophyseal function through atrophy of the anterior lobe (hypopituitarism). See also Simmonds.

The hypophysis is an organ necessary to life. After total removal (Cushing, Biedl, lit.), older animals die in one to four days, younger animals in one week, while some develop a cachexia hypophyseoprivia and finally die in coma. These experiments have been confirmed by

observations in humans. As Simmonds first pointed out, disappearance of the hypophyseal function (anterior lobe) in later age (apituitarism) leads to cachexia with anemia, nervous symptoms, disappearance of the genital function, and finally quick death in coma. Pathological changes in the glandular part of the hypophysis include necroses, tuberculosis, gummata, basophilic adenoma, connective tissue atrophy; according to Budde (lit.), a hyperfunction such as occurs during pregnancy may be followed by atrophy. See also E. Fraenkel, Schlagenhaufer, Fahr.

Degenerative changes are quite frequent. There occur: cystic colloid changes of the glandular elements, pigment degeneration, cystic softening of the stroma, the development of numerous blood vessels in the stroma, amyloid infiltration of the blood vessels, and laminated calcium concrements. Toxic necroses occur in diphtheria; experimentally they have been obtained by Creutzfeldt and Koch, by the injection of diphtheria bacilli. Circulatory changes and embolic processes, metastatic inflammations, etc., may also be found; hemorrhages following trauma have been reported, as well as necrosis due so arteriosclerosis or embolism, such as in verrucose endocarditis (Merkel). Scars and fibrous atrophy of the hypophysis may result from necroses (Simmonds). That metastatic embolic processes are quite common have been shown by the investigations of Simmonds. Bacterial emboli in the posterior lobe lead to small abscesses, in the anterior lobe to embolic necroses (anemic infarcts). Symptoms of lack of function may follow embolic damage. In thirty-five cases of pyemia, A. Plaut found hypophyseal foci in seventeen, particularly in infections with the staphylococcus.

Tuberculosis occurs in two forms. (*a*) The hematogenous is more common in the anterior lobe; it is found in general miliary tuberculosis, even in children, and also in pulmonary tuberculosis; occasionally, larger solitary caseous nodules appear, more often in women. (*b*) Tuberculosis by direct extension comes from the meninges, or from tuberculosis of the bone. Diabetes insipidus may finally occur (p. 1949). (Lit. by M. B. Schmidt, Hueter, and Zenoni; for destruction of the hypophysis without acromegaly, see Heidkamp, Simmonds, lit., Schmorl, Froboese; primary isolated tuberculosis, Schmittmann.) Destruction of the hypophysis leads to fatal cachexia. In tuberculosis of the sphenoid bone with involvement of the hypophysis, Kurzak (lit.) saw symptoms of hypophyseal hypofunction. In a few rare cases in women beyond their fiftieth year, Simmonds found giant cells of Langhan's type in the anterior lobe without tuberculosis, and without tubercle bacilli, which possibly were called forth by the irritation of abnormal secretion.

Syphilis has been observed as a diffuse interstitial inflammation, or as miliary gummata in congenital syphilis (Simmonds, Schmorl). Larger

gummata in the anterior and middle lobes in adults are very rare (Stroebe, lit.). (Lit. by Froboese.) Schafer saw hypophyseal cachexia after gummatous destruction. For more details, see Nonne (lit.).

Pineal Gland, Epiphysis

Anatomy: the pineal gland appears first from the diencephalon, as a projection like the finger of a glove; this soon bends backward, and comes to lie free in the folds between the anterior corpora quadrigemina. The anterior fixed part retains a small cavity communicating with the third ventricle, called the recessus pinealis, whereas the posterior part becomes a compact gland-like body (for more details, see Broman). This gland has an average weight of 0.157 gram (Uemura).

Histology: (consult Krabbe, Schlesinger, Uemura). The pineal gland is composed of (*a*) gland cells, (*b*) glia cells, (*c*) nerve cells, and (*d*) connective tissue. The gland cells are arranged in irregularly shaped solid alveoli, which comprise the main mass of the parenchyma. They are rounded or polygonal, poor in protoplasm, with nuclei rich in chromatin. Peculiar to the latter are the "nuclear granules," *i.e.*, basophilic granules which extend from out the nucleus into the protoplasm. (*b*) Glia cells of the fiber-forming type are not important. (*c*) Nerve cells with chromatin-rich nuclei are likewise an unimportant part; the prolongations of (*b*) and (*c*) form a fibrillar network in which the gland cells are embedded. (*d*) The mass of connective tissue varies; in children or older people it is usually small in amount, but the reverse also occurs. It comes from the adventitia of blood vessels. Connective tissue septa are connected with a fibrous capsule. The gland may also contain pigment cells. Acervulus cerebri, or brain sand, composed of mulberry-shaped concrements (p. 1818), is found from the sixth to the seventh year onward; the amount varies; in adults it is larger; larger collections produce psammoma-like structures.

In adults, we observe involution changes. The parenchyma becomes less in amount, globular, and surrounded by the dominant connective tissue. The latter forms broader septa, shows hyalin degeneration and calcification which may lead to induration. For more details in histology, see Josephy. But there always remain, even in old age, individual variations in the amount of parenchyma (Krabbe, Schlesinger). Uemura saw calcification of the interstitial substance, the blood vessels and the parenchyma, even in a four and one-half year old child. Cysts are quite common in adults, due, it is thought, to focal softening of the glia, (for other views, see Krabbe). They have been seen in children.

Pathological Anatomy.— Cysts have been mentioned above; if quite numerous, the condition is known as hydrops cysticus glandulae pinealis.

There also occur cysts of the recessus pinealis and ependyma cysts in the basal part. Psammoma-like calcium collections have been mentioned above. In suppurative meningitis, abscess formation may occur in the pineal gland. Tuberculous changes are rare. In congenital syphilis, there has been observed diffuse infiltration with miliary gummata; gummatous changes are quite rare in acquired syphilis. Uemura saw hypertrophy of parenchymatous character in adiposity. Primary tumors, occasionally as large as hen's eggs, are rare, but very important. Metastatic tumors such as sarcomas (Jacobi) and carcinomas, (lit. by Askanazy), are extremely rare. Among the primary tumors, there have been described: by far the most frequent, sarcomas, among them round cell, alveolar, fibrous, psammomous; these sarcomas, however, are not always

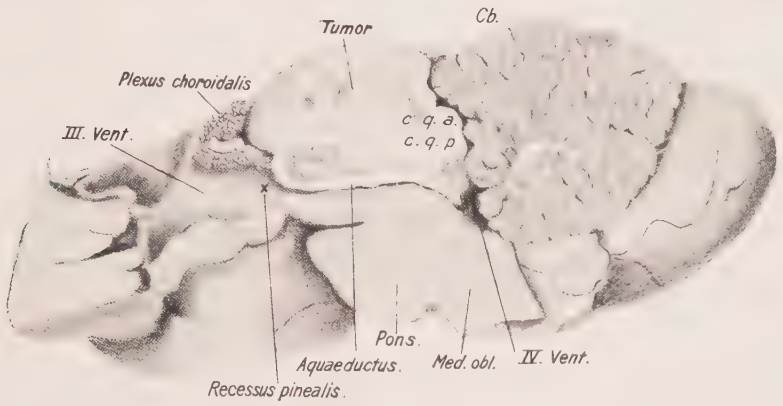


FIG. 956.—Tumor of pineal gland (so-called compound tumor, after Marburg). Pappenheimer, V.A., 200, 1910.)

of one kind of tissue; thus, Marburg believes some of them are "mixed tumors," composed of pineal gland tissue, ependyma cells and glial cells; furthermore, they have been described as dermoids and teratomas (Fukuo, lit.), particularly in males, usually in younger individuals. Adenomas, as well as tumors regarded as carcinomas, gliomas, neuro-glioma ependymale, etc., have all been reported. (Hart, Pappenheimer, Boehm, lit. by Marburg, by Berglinger, and by Giebel).

In a young man nineteen years old, Askanazy found a chorion epithelioma which he believes equivalent to an embryonal teratoma (compare p. 1747); a similar tumor was described by Goldzieher. Others believe they were angioblastic sarcomas.

Physiology.—The pineal is a gland of internal secretion (Marburg). It is not a rudimentary organ, nor is it probable, according to Schlesinger, that there is a simple atrophy in old age. Perhaps the secretion has an influence on the genital glands, by preventing their complete development

until the time of puberty (Biedl). In fact, the pineal has been called the "antipuberty gland" (Posner), and there has been postulated an antagonism between the pineal gland and the interstitial cells of the testes; the latter, according to Posner, comprise the actual so-called "gland of puberty;" the secretion of the antagonist reduces potence and libido. But all of these ideas are merely hypotheses. The experimental investigations of Exner and Boese, in which they extirpated the pineal gland in young rabbits, showed no influence on the growth and the appearance of puberty. On the other hand, extirpation experiments of Horrax in guinea pigs, and particularly in rats, showed a hastened development of the sex organs in the male, while C. Foa in experiments on roosters saw a marked hypertrophy of the testes and of the comb eight to twelve months after the extirpation of the pineal body. Following castration in cats of of both sexes, Sarteschi found atrophy of the pineal, and hypertrophy of the hypophysis.

Pathological-physiology.—The results of tumors of the pineal have afforded the chief sources of information concerning its physiology. There has been observed the rare coincidence of pineal gland tumor (usually teratoma), and gigantism (Ostreich-Slawyk), in which there was, of course, not a typical acromegaly, but an hypertrophy of the genitals, premature development of sex (pubertas praecox, large, erectile penis in small children and even ejaculation), and early appearance of hair on the pubes and in the face; this symptom complex, found exclusively in children, and called "macrogenitosomia praecox" by Pellicci, may be coincident with early changes in the voice, early psychological sex feelings, and occasionally with adiposity. (Lit. by Marburg, v. Franke-Hochwart, Askanazy, E. Boehm, Lowenthal.) Opinions vary as to the exact relationship in these conditions. Is the early puberty of pineal origin, *i.e.*, from disappearance of the pineal gland (Marburg)? Is it oncogenic, *i.e.*, caused in some of the cases by the embryonal tissue character of the tumor (Askanazy), which chemically influences the organism as a whole? Is the fact that the majority of cases occurs in boys due to the relatively greater frequency of these tumors in boys, or is it due to a special function of the male pineal? These are questions which remain to be answered. (Consult Marburg, and see below.) The case of Askanazy and Brack (lit.) of sexual praecocity in a microcephalic idiot with hypoplasia of the pineal is of great interest; it seems to indicate that the function of this gland is the same in both males and females; the case is the more valuable since the other classical organs of internal secretion, the hypophysis, adrenals, pancreas, were unchanged, and a change in the thyroid could not be brought into relationship with this precocity. Others believe there is a correlation between the sex glands,

the hypophysis, and the pineal, and an antagonism between hypophysis and epiphysis (Munzer, lit.); disappearance of the function of the pineal leaves the hypophysis in a position to act without inhibition on the development of the sex glands, and to bring forth an early development of puberty (Goldzieher, lit., and Askanazy). In their case of a ten year old boy with a cystic glioma of the pineal gland, Raymond and Claude found adiposity and "smallness" of the genitals, *i.e.*, atrophic testes, with, however, well developed interstitial cells! This case belongs rather to the symptom complex of hypophyseal tumor; it was accompanied by growth of hair and early psychological sexual awakening; they postulate that other internal secretory glands complicated the picture. In this case, Marburg believes there was hypersecretion of the pineal gland which led to adiposity, in contradistinction to apinealism which leads to severe cachexia,—but in the animal experiments mentioned above, cachexia never occurred!—and that a hyposecretion in contrast to apinealism, leads to early development of the sexual function. With E. Boehm, the author doubts that a glioma with cysts which occupied the situation of the pineal gland could bring about an oversecretion of the latter structure. It must be remembered that cerebral symptoms are met in tumors of the pineal gland, such as hydrocephalus from pressure of the tumor on the vena magna Galeni and the aqueduct, with hydrops of the third ventricle, and with paralyses of the muscles of the eye. The mixed symptom complex in the case of Raymond and Claude is perhaps better explained by marked hydrocephalic pressure on the hypophysis leading to a mixed hypophyseal and epiphyseal effect. There is no doubt that the function of the pineal gland and its correlation with the other internal secretory organs require much more experimental work. This is also true of the relationship of the pineal to lipodystrophy, *i.e.*, disappearance of fat in the upper parts of the body in later childhood, also fat hypertrophy in the pelvis, the gluteal region, and the calf of older female individuals (Klein, lit.). Other literature in the appendix.

SPINAL CORD

SPINAL MENINGES

Anatomy of the Spinal Meninges.—The tough, thick, dura mater spinalis is anatomically different from the similar structure in the skull; it forms a sac separated by the so-called peri- or epidural space from the bony spinal column which is lined with periosteum; it also does not rest tightly upon the spinal cord (figs. 957 and 958). In the epidural space are tender connective tissue, fatty tissue, and many blood vessels, particularly venous channels. (This red connective tissue mass has often been

considered pathological, and the seat of inflammatory changes.) Opposite each opening in the intervertebral discs, the dura has two openings through which pass the corresponding spinal nerves, (fig. 958). The dura surrounds these nerves like a tube and then becomes lost in their sheaths. It is fastened outside each opening by fine, fat-containing connective tissue. The inner surface is covered with a simple layer of flat endothelial cells. In old age, calcification of the dura, at least in microscopic amounts, is quite frequent; occasionally there is macroscopic calcification posteriorly, *i.e.*, where the dura surrounds the vertebral arches (Heschl and Ludwig; see also M. B. Schmidt).

The **relations of the soft membranes,** the arachnoid and the pia, are similar to those in the brain. They comprise a sac with two leaves, which form the subarachnoid space containing the cerebrospinal fluid.

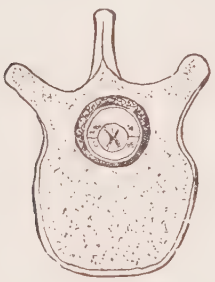


FIG. 957.

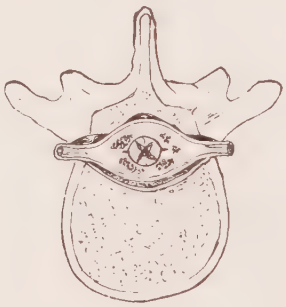


FIG. 958.

FIG. 957.—Horizontal section through X thoracic vertebra.

FIG. 958.—Section through first lumbar vertebra with exit of XII dorsal pair of nerves. In both figures the spinal cord, covered by pia, is in the center, being held on both sides by the denticular lig.; the nerve roots form bilateral groups. Between dura and cord, and between dura and wall of vertebra in each case is a wide space, the latter filled with vascular connective tissue; no arachnoid sketched. After Key and Retzius.

The outer leaf, the arachnoid, is not attached to the dura; the outer surface of the arachnoid and the inner surface of the dura are separated by a space clothed with flat epithelial cells. It is only in the cervical region that under normal conditions there are fine adhesions between the dura and the pia-arachnoid, (these must not be confused with inflammatory residues). The pia, which is closely approximated to the spinal cord, sends a thick vascular connective tissue prolongation into the anterior median fissure of the spinal column (septum medianum anticum). Vessels surrounded by lymph sheaths enter the spinal cord from the pia. The arteries of the spinal cord are the anterior spinal and posterior spinal from the vertebrals, and a number of smaller vessels from the intercostal, the lumbar, and the sacral arteries. Anastomosis between the blood of the vertebral arteries and the intercostal arteries is accomplished through the so-called vaso-corona, a network of vessels surrounding the periphery

of the spinal cord; the intercostal arteries come mostly from the aorta (ninth to the tenth branches); the first two to twelve posterior intercostals arise from the subclavian arteries. The intercostal arteries from the aorta follow the spaces and the functions of the lumbar and sacral arteries. The part most poorly supplied by vessels is the lower thoracic cord; see the experiment of Stenson, ligation of the aorta. Compare also page 145, paraplegia after tearing the intercostal arteries in an dissecting aneurysm.

The spinal cord has an average length of forty-five centimeters, a weight of thirty-five grams.

Diseases of the Dura Mater Spinalis

Circulatory Disturbances.—Hemorrhage into the epidural tissue occurs after trauma to the spinal column, in asphyxia, particularly in the newborn, and also in tetanus, like the hemorrhages occurring in animals poisoned with strychnine. Usually the blood is coagulated between the periosteum and the dura.

In the newborn, it is observed both after Schultze's method of resuscitation, and in 10% of other operative births, and other birth injuries (O. Schaffer); hemorrhages may also occur under the pia and in the spinal cord itself (G. Burckhard). Care is necessary in judging whether intrauterine asphyxia alone can produce the anatomical picture which may be blamed on Schultze's method of resuscitation (compare B. S. Schultze, and opposed Hengge).

Inflammation.—Acute and chronic inflammations are usually extensions from the outside (pachymeningitis externa), *e.g.*, from caries of the vertebral column, abscesses or bed sores in the sacral region (in paraplegia, etc.), bed sores over spina bifidas, or traumatic opening of the vertebral canal. The inflammation is usually suppurative; it may be focal or diffuse. Occasionally, the amount of pus is sufficient to compress the spinal cord. The internal surface of the dura may also be involved (pachymeningitis interna).

Chronic inflammations occur:

(*a*) As a pachymeningitis interna hemorrhagica. This is either an accompanying symptom of a specific inflammation of the spinal meninges, or is independent, as in cerebral paralyses, in inebriates, when it is analogous to the pachymeningitis interna hemorrhagica of the cerebral dura. The condition occasionally involves both spinal and cerebral dura.

(*b*) In pachymeningitis cervicalis hypertrophica (Charcot-Joffroy), there appears a laminated, onion peel-like, thick, cicatricial connective tissue formation, first in the lower part of the cervical region on the inner surface of the dura (pachymeningitis interna fibroplastica), and this membrane reaches five to ten times its normal thickness.

This thick connective tissue layer, which may even ossify, usually spreads annularly around the spinal cord in the cervical region, may reach a length of nine centimeters, but be so circumscribed, particularly, and most frequently, in the lower part of the cervical region, that it gives the impression of a fibrous tumor. The soft membranes also take part in the productive inflammation, and it enters the spinal cord through the septa and the chronically inflamed vessels. Indeed, many believe that the process is primary in the soft meninges, and that these are the source of the connective tissue masses formed between the arachnoid and the dura. Finally, the dura and the soft meninges with the nerve roots and the spinal cord itself, disappear in a cicatricial mass. At first, there is irritation, then atrophy of the nerves and sclerosis of the compressed spinal cord, or even total transverse atrophy.

Etiology.—Exposure to cold, trauma, but particularly syphilis, have been held responsible; many believe that the latter disease is the most frequent cause. Course: the duration may be years; occasionally the process is interrupted, but healing rarely takes place. Among the clinical symptoms may be mentioned: (1) the stage of neuralgia, particularly in the upper extremities, *i.e.*, the stage of irritation of the roots; (2) the stage of anesthesia, paralysis and atrophy in the sensory and muscular regions of the compressed nerve roots, particularly the ulnar and median nerves; "main en predicateur" appears because of the greater strength of the extensors; (3) in the third stage, the spinal cord is also involved. Later, when an entire transverse section is compressed and the spinal cord destroyed, the symptoms of transverse myelitis occur. The disease may not, however, progress this far, but may even heal, of course, with defects. According to v. Leyden-Goldscheider, Charcot's disease is not a clinical entity produced only by this type of meningitis, but is rather a meningomyelitis cervicalis caused by different conditions such as spondylitis or tumors, (B. Schultze; Wieting).

Infectious Granulation Tumors.— **Syphilis** leads to diffuse fibrous thickening of the dura and to adhesions between this structure and the other membranes. More frequently, however, the point of origin is in the soft meninges (meningomyelitis luetica), with invasion both into the substance of the spinal cord and into the inner surface of the dura, a picture quite similar to that of pachymeningitis interna chronica (Charcot). Occasionally, but much less often than in the cerebral dura, there are gummatous overgrowths (lit. Nonne). Tuberculosis of the dura as a chronic inflammation of the outer surface is seen most frequently in caries of the vertebral column (see p. 1130, Pott's disease). Not rarely, the outer surface of the dura is covered over long stretches with a caseous mass almost like a tumor, to the thickness of a finger. In the dura itself are

caseous masses which may compress the spinal cord. It is rarer for the tuberculous process to penetrate the dura and establish itself on the inner surface; from there, however, a miliary tuberculosis may spread to the dural cavity, or a specific internal pachymeningitis may be produced. An isolated tubercle of the spinal dura is unique, (Baumann).

Tumors and Parasites.—Primary tumors are rare. They may be fibromas, particularly fibroendotheliomas (fig. 959), which eventually become psammomatous; they are often lateral and ventral; older individuals are preferred (Antoni); they occasionally surround the spinal cord with an egg-shaped or cylindrical mass; again, they become quite large, or are multiple, in which event they are usually small (Troitzky, Henschen). Furthermore, there have been described: myxomas and sarcomas, fibro-psammo- or melanosarcomas (the so-called melanomas or chromatophoromas; Boit, Pol, Thorel), also cylinder-like sarcomas with hyalin changes of the vessels (Kawashima). These tumors may be partly extra- and partly intradural; in both cases they are spoken of as extramedullary, intravertebral tumors, to which belong also the tumors of the leptomeninx and the vertebral column. Sarcomas may arise from the peripheral external parts of the dural sac, or, just as in the skull, from the inner surface; they are usually solitary ovals as large as hazel nuts (for the literature of these tumors, which are often surgically remediable, see Flatau); in other cases there is dissemination in the meningeal space, and a secondary spread into the cerebrospinal pia (Kawashima, lit.). Diffuse overgrowth of fat in the epidural space is seen in connection with chronic inflammation of the spinal column and cord, and its membranes. Very rarely, there are distinct lipomas, usually toward the caudal parts. Teratomas (congenital) with cartilage and connective tissue, among other things, occur in the sacral and coccygeal regions; they are usually medial.

Fig. 959.—Broad based (intradural) partly psammomous, fibro-endothelioma of the spinal dura, (3 cm. long, 2 cm. thick) at right ventral periphery of level of 4 to 6 dorsal nerves. The tumor is cut open lengthwise. Deep impression into cord. Transverse myelitis. Woman 48 years old.

Secondary tumors are much more frequent, and of more practical importance; they appear externally or internally on the dura, or more frequently, they invade the dura directly from the exterior; the latter are from the vertebral column, and are more common than all other menin-

geal and medullary neoplasms (Schlesinger). The usual source of these metastatic vertebral column tumors is carcinoma of the breast or other organs; sarcomas are rarer (Guleke), as are chondromas, osteomas,—which come from the vertebrae, or from the neighborhood, and have invaded the vertebrae.

Carcinomatous masses are particularly prone to fill the vertebral canal, so that the spinal cord is directly compressed or compromised as a result of disturbances in the circulation of the blood and the lymph, whereupon extensive paralyses appear. Severe nerve pains are the result of compression, or growth, especially into the posterior roots, and are often present in paralyzed members. Not rarely, the vertebrae, infiltrated by tumors, undergo fracture, most frequently from secondary carcinomas. But fracture of merely one vertebra may also occur, as *e.g.*, because of an angioma, which may spread epidurally at the same time (Muthmann). The spinal cord is then crushed into an acute, so-called compression myelitis, which usually leads to transverse paralysis (fig. 978). Auerbach described an extradural chorion epithelioma which appeared nine months after abortion, and which led to compression myelitis.

Parasites.—Cysticerci in the dural sac are very rare; they rather appear from cysticercosis of the base of the brain as free swimming parasites in the vertebral canal, (Furnrohr). Echinococci have been frequently observed, sometimes in the dural sac, when they are rounded, or egg-shaped, and compress the medulla, sometimes, and more frequently, extradurally. They are usually secondary to those in the muscles about the vertebral column, or in the subpleural or retroperitoneal connective tissues, and very rarely to the vertebral bodies themselves. If they force their way from the exterior through the intervertebral spaces into the vertebral canal, they form tumor-like structures, which lie both outside and inside of the vertebral column like sand glasses, connected to each other by a sort of pedicle (compare sarcomatous hour glass-like tumors of the vertebral column, Guleke).

Diseases of the Spinal Pia-arachnoid

Circulatory Disturbances.—Hemorrhages may appear in the subarachnoid space after trauma, in inflammations, in the hemorrhagic diatheses, in the agonal stasis, following cerebral hemorrhages, as well as from unknown causes. Larger hemorrhages (apoplexia canalis spinalis) are found as larger blood coagula, particularly around the nerve roots.

See Gaupp, Lindemann, concerning the formation of varices in the vessels of the pia and spinal cord as causes of a total transverse lesion.

Inflammation (Meningitis Spinalis).—Acute inflammations follow traumata, or direct extension from neighboring parts, *e.g.*, a sacral decubi-

tus, or by way of the blood stream. The exudate may be seropurulent, purulent, or fibrino-purulent, and thus watery, viscid, or creamy; it infiltrates the pia and collects in the subarachnoid space. The organisms are usually the ordinary pyogenic cocci.

Another form of spinal meningitis, which usually involves both the spinal and cerebral meninges, and which often appears in epidemic form, has been described under the brain; in typical cases, the specific microorganisms are found (p. 1830), with inflammatory edema, round cell infiltration, or focal softening. As a rule, the bordering parts of the spinal cord and the nerve roots are also affected. The condition is therefore called meningo-myelitis and -neuritis.

Extension into the cerebral meninges in traumatic suppurative meningitis is often quite rapid, *e.g.*, after fracture of the vertebrae, gun-shot wounds, operations, or spontaneously from spina bifida, or decubitus. Occasionally, the entire cerebrospinal subarachnoid space and even the ventricles of the brain are filled with creamy pus in as short a time as twelve to twenty-four hours.

Chronic leptomeningitis spinalis may, as stated, be combined as a meningo-myelitis with a pachymeningitis chronica hyperplastica (p. 1961). Furthermore, white, opaque thickening of the meninges, occasionally with adherence of the dura, is not infrequent over carious spots in the vertebral column. These same milk-white thickenings of the soft membrane follow traumata and intramedullary conditions which spread peripherally. Local thickenings are found occasionally in syphilis.

Meningitis serosa chronica circumscripta or adhesive circumscribed arachnitis is of practical importance because its pseudo cysts—encapsulated collections of cerebrospinal fluid—clinically simulate the pressure symptoms of tumors; they follow traumata, hemorrhages, etc., (Oppenheim and Krause); the condition is not influenced by lumbar puncture; it may also appear in similar guise in the brain (p. 1825, Krause, Oppenheim, lit., Grossmann, Bonhoeffer, Gerstmann, lit.). See Mauss and Kruger for war injuries simulating meningitis serosa chronica circumscripta.

Infectious Granulation Tumors.—Tuberculosis (1) may spread from the dura, or from the vertebrae, or (2) may be hematogenous, or (3) lymphogenous; the latter is particularly frequent in tuberculous meningitis (p. 1834). It occurs more frequently in the thoracic than in the cervical part. As a rule, the dorsal surface of the cord is more markedly involved. Occasionally, it assumes an almost nodular condition. Very often the tuberculous meningitis is accompanied by peripheral myelitis which appears as a pale or red softening over larger areas; hemorrhagic infarction may be produced by thrombosis; the roots, as well as the nerves, may

be involved (tuberculous meningo-myelitis and -neuritis). Nodules in the spinal cord itself are rare.

Lymphocytes predominate in the centrifugalized specimens of lumbar puncture fluid.

Syphilis.—Leptomeningitis, or better meningo-myelitis syphilitica, leads to gray, or gray red, bacony or gelatinous granulations, which later become flat or plaque-like, gray white, or yellow (necrotic) tough thickenings. The blood vessels are always involved, sometimes markedly so, as an endarteritis or a phlebitis obliterans; compare p. 1918. The varying symptoms of the disease are determined by the focal degenerations in the cord due to the localized involvements of the blood vessels. Syphilitic meningo myelitis spinalis may be combined with basal cerebral meningitis (lit. by Boettinger, Meyer, Scheiber, and particularly Nonne).

Leprosy.—There occurs a leprous meningitis, with the formation of a gray or gray yellow exudate, established by preference on the posterior circumference of the medulla and around the nerve roots; it is usually confined to the spinal meninges.

Tumors of the Soft Spinal Meninges. (Lit. in Appendix.)—In over half of the individuals over forty years of age, there are found spicules of

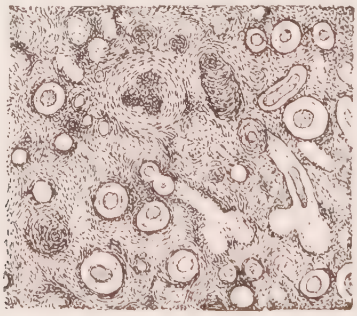


FIG. 960. FIG. 961.

FIG. 960.—Hemangiosarcoma with hyalin degeneration of vessel walls, from tumor fig. 836. (Low power.)

FIG. 961.—Sarcoma of spinal arachnoid, transverse; above, tumor; below, light spinal cord; outside fragments of dural sac; nat. size.

bone, small osteomas, in the arachnoid, usually on its posterior surface. They are white, porcelain-like, irregularly constructed, externally smooth, internally rough. They are adherent to the pia, particularly in the region of the cauda. They arise by calcification and ossification of a hyalin or fibrillar substrate and become vascularized from the dura (Zanda). Occasionally they are osteoid. They have no pathological significance, produce no symptoms, nor are they a manifestation of an earlier trauma.

As primary tumors, there occur: fibromas, hemangiomas (lit. by Lindemann, rare), myxomas (very rare), sarcomas, particularly the round

cell variety, also the rarer forms such as angio-sarcomas with hyalin degeneration (the so-called cylindromatous formation), (see figs. 960 and 961), alveolar endotheliomas, which, arising from the endothelial covering of the arachnoidea, are sometimes psammomatous fibro-endotheliomas (lit. Stursberg, Hertz), peritheliomas (Lissauer); there also occur melanotic sarcomas (p. 1844), which may spread diffusely or in spots. Lipomas have been described, but very seldom (Meyerhoff, Ritter, lit.), as well as myolipomas at the caudal end of the cord—a congenital tumor combined with spina bifida. In the nerve roots, multiple neurofibromas (fibro-spindle cellular neurinomas) are frequently found as part symptoms of the disease, neurofibromatosis; they may become as large as walnuts; these have also been met in the bundles of the cauda equina.

See Henneberg, concerning tumors of the posterior furrow of the spinal cord (hemangiomas, lipomas, teratomas).

As secondary tumors, there have been described carcinomas and sarcomas and even gliomas of the retina, which, following the sheath of the optic nerve, have reached the base of the brain; these secondary tumors appear as direct extensions or by metastasis (seldom), particularly in melanotic sarcomas. There may be differentiated: (*a*) pure carcinomatosis from (*b*) a fibrino-serous or a suppurative carcinomatous meningitis. The former may lead to compression of the spinal cord. There also occur cases (*c*) of metastatic carcinosis of the meninges which can only be recognized microscopically, and which lead to nothing but mild cloudiness and thickening like a chronic leptomeningitis, or which appear like an edema, or a suppuration. See Eichhorst (lit.), also Lissauer, concerning this "anatomically latent" meningeal carcinoma. In a case observed in Breslau, the primary tumor was a sarcoma in the dura of the cerebrum, and there were innumerable small metastases in the pia-arachnoidea along the entire course of the subarachnoid space of the spinal cord; they had localized particularly in the lower part, around the fibers of the cauda, which was surrounded by tumor masses in the form of rounded and spindle shaped nodules. In such a case, it is perhaps justifiable to think of "vaccination" of tumor cells. Similar pictures are sometimes seen in carcinomas. Occasionally, the secondary infiltration is very diffuse (lit. by Nonne); there occur other cases, however, in which the metastases are diffuse from the very beginning (lit. by Rindfleisch). For the diagnosis by lumbar puncture, see p. 1844.

See Strassner (lit.), concerning diffuse extramedullary spread of gliomas, which usually is secondary to a primary tumor in the brain or the spinal cord (Rutimeyer, lit.), but may also come through leptomeningitis, and, as the author saw, from the nerves of the cauda equina which become thickened to nodular, varicose strands. In a glioma of the cervical

part of the spinal cord, Mees saw only "regional metastases" of the surrounding meninges.

Most of the tumors of the soft meninges grow slowly, and are small; they occasionally become larger when they spread in the longitudinal direction and take the shape of eggs, or fingers, and become quite thick. There even occur primary sarcomas which spread diffusely along the entire spinal cord. The meningeal (mostly pial) tumors often compress the cord without producing any other changes within the structure; in other cases, however, they gradually cause compression symptoms of the nerve roots and the cord itself. More rarely, the tumor invades the spinal cord. Etiologically, traumata are often blamed, particularly for sarcomas.

In meningeal as well as in actual spinal cord tumors, a yellow discoloration of the spinal fluid (xanthochromia) is frequently observed; it is supposedly due to previous hemorrhages (Grund), but, as a rule, the blood coloring matter cannot be identified spectroscopically, (for exceptions, see Geissler). There may be coagulation and lymphocytosis at the same time, (Frouin; compare Klieneberger, Siemerling, Rutimeyer, lit.).

Spinal Cord

Preliminary Remarks.— These are made merely for orientation of the gross anatomic relationships. Many details concerning the finer structures will be mentioned in appropriate places in the text.

The spinal cord does not stretch along the entire spinal canal, but ends in the conus terminalis somewhere in the neighborhood of the lower edge

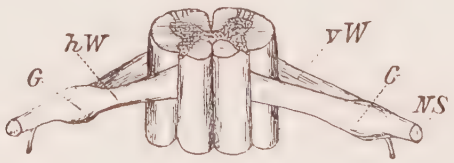


FIG. 962.—Segment of spinal cord with its pair of spinal nerves (modified after Bramwell). vW, anterior root; hW, posterior root; G, ganglion of posterior root; NS, cross sect. of trunk of spinal nerve (mixed).

of the first lumbar vertebra. In the lower portion of the spinal canal, there lies the cauda equina, a bundle of spinal nerves, the roots of the lumbar and sacral cord, which, at first, run for varying lengths in the spinal canal vertically from above downward, until they reach the foramina intervertebralia of the lumbar vertebral column and the sacral foramen. The segments of the spinal cord vary in their thickness at different levels. The cervical swelling lies near the level of the third and fourth cervical vertebrae and reaches its maximum at the fifth and sixth vertebrae. The thoracic cord begins at about the level of the second

dorsal vertebrum, and remains about the same thickness throughout. The lumbar swelling appears in the lumbar cord at about the tenth to the twelfth dorsal vertebrae. The sacral part extends from here to its end in the conus terminalis at the first to the second lumbar vertebrae.

The spinal cord is composed of an external or cortical part (white matter) and an internal part (gray substance). On cross section, the gray matter is in the shape of the letter "H;" in front and behind it shows two prolongations, the anterior and posterior horns; the former are blunt, the latter, pointed. The transverse part of the "H" is formed by the gray commissure, which contains the central canal lined with cylindrical epithelium. The anterior or motor roots emerge from the anterior horns, and the posterior or sensory roots from the posterior horns. The posterior root belongs to the ganglion intervertebrale (spinal ganglion, a collection of ganglion cells; see Dogiel concerning the more detailed structure and their various types). A spinal nerve (mixed) results from the union of the anterior and posterior roots. A segment of spinal cord is a slice of nerve tissue belonging to a pair of symmetrical spinal nerves.

There are thirty-one pairs of spinal nerves, eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal. The number of the spinal nerves does not correspond to the number of the vertebrae from which they appear. With the exception of the upper cervical region, their origin is usually higher in the cord than their place of exit from the vertebral canal. The distance between them becomes much greater from above downward. Thus, for example, the seventh cervical vertebra is the point of exit of the first dorsal nerve; the spinous processes of the fifth and the seventh thoracic vertebrae are the points of exit of the seventh to the ninth dorsal nerves. The first lumbar nerve emerges at the region of the eleventh cervical vertebrum; the second lumbar nerve between the eleventh and twelfth; the third and fourth lumbar nerve in the neighborhood of the twelfth thoracic vertebrum; the fifth lumbar nerve and the first sacral between the twelfth cervical and the first lumbar vertebrae; the second to the fifth sacral nerves at about the region of the first lumbar vertebra; naturally, they have a longer course to pass through in the vertebral column the deeper their point of exit from the sacrum.

The white substance is divided into anterior, lateral, and posterior strands by the spinal roots and the gray substance (fig. 963). The white substance is composed chiefly of medullated nerve fibres gathered together in strands. The arrangement of these strands into bundles (specific paths) cannot be identified in the spinal cord of the adult. Their recognition has been partly due to embryological studies (Flechsig), inasmuch as the single bundles do not come to possess medullary sheaths at the same time. Pathological changes of isolated bundles and paths have given an insight

into their complicated arrangement; particular systems of fibres become diseased and can thus be differentiated.

The various paths or systems of fibres in the white substance are as follows (fig. 963):

1. In the anterior strands on each side.

On both sides of the anterior longitudinal fissure are the pyramidal tracts, (*a*) or Turk strands, or the direct uncrossed pyramidal tracts, which connect the motor centres in the cortex with the gray substance of the anterior horns, and then with the muscles. The path becomes smaller as it descends, and almost disappears in the middle of the dorsal cord.

The remainder of the anterior strand is composed of the anterior basal fasciculus (*b*).

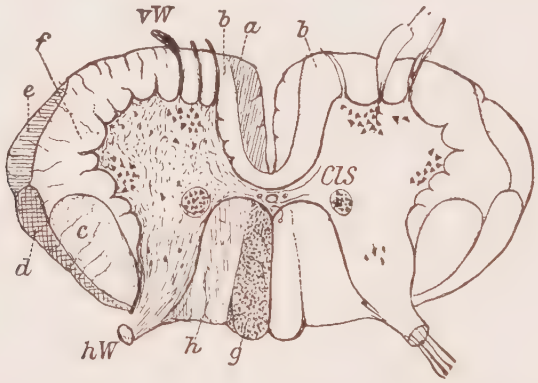


FIG. 963.—Cross section of cervical enlargement of sp. cord; l. half. a, anterior pyramid tract; b, anterior ground bundle; c, pyramidal lateral tract; d, cerebellar lateral tracts; e, Gower's tract; f, lateral ground bundle; g, Goll's column; h, Burdach's column. To right, only outlines of corresponding tracts; ClS, Clarke's column; vW, anterior root; hW, posterior root; diagramatic.

2. In the lateral columns on either side.

The lateral pyramidal tract, (*c*) or the crossed pyramidal tract in the posterior portion. They are a part of the motor or the cortico-muscular path; they contain motor fibres (centrifugal) which gradually pass into the gray substance; the fibres split into fibrils, and surround the cells of the anterior horns. The impulses are then carried through contact to these ganglion cells, through the intermediation of the fibrillar connections, as mentioned on p. 1990. From the anterior horn cells there then appear the motor anterior roots. On cross section, the lateral pyramidal tracts assume the contour of a triangle; in the cervical and thoracic parts, they do not reach to the edges as they do in the lumbar cord. They are largest in the cervical cord, but become smaller from above downward (fig. 973).

The lateral cerebellar tracts (*d*) lie peripherally in the posterior part, and contain fibres which enter from Clarke's columns, and course upward (centripetal) to the superior vermiform process; this path, which on cross

section has somewhat the contour of a sickle, begins first in the lower thoracic cord and becomes larger as it ascends.

Gower's bundle (fasciculus antero-lateralis ascendens) (*e*) lies in the anterior lateral periphery. This carries sensory nerves coming from the gray matter and begins in the lumbar cord.

The remainder consists of the mixed lateral parts (*f*).

3. In the posterior strands on either side:

The middle part is composed of Goll's strand or the funiculus gracilis (*g*), a sensory path.

The column of Burdach, or the funiculus cuneatus (*h*), likewise a sensory path, separated from the column of Goll, at least in the cervical part by the septum paramedianum, occupies the lateral region.

(For more details concerning the division of these sensory fibres in the various heights of the cord, see under the heading "ascending degeneration" p. 1984.)

Malformations of the Spinal Cord

See p. 1977 for hydromyelia or hydromyelus, hydrorrhachis interna' congenital dilatation of the central canal by abnormal accumulation of cerebrospinal fluid. A circumscribed hydromyelus is called myelocyst. Hydrorrachis externa is a collection of fluid between the membranes, particularly in the subarachnoid space. Reduplication of the central canal is rare. Reduplication of the cord (diastematomyelia) occurs principally because symmetrically placed parts of the spinal cord on each side fail to unite; they are therefore fissures; at times, there do occur true double structures; reduplication of the central canal is then the causative factor (Zingerle, experimental observations by Waelsch). With the exception of a case of Westphal, they are combined with spina bifida lumbalis or cervicalis, generally with a cystic, and seldom with an occult form (Zalewska-Ploska, lit., Henneberg and Westenhofer, lit., Henneberg, lit.); occasionally they are combined with other malformations. Only very rarely does the reduplication involve the entire length (von Monakow, lit.). Great caution must be observed not to confuse these and other actual malformations with artefacts (van Gieson).

Fissure of the spinal column, rachischisis, occurs throughout a part or its entire length; it is caused by failure of union of the bilateral anlage of the vertebral column. In total rachischisis, we usually look upon a thin, transparent membrane, the inner surface of the ventral part of the pia, and under this the dura lies upon the flat edges of the vertebrae. Rudiments of the spinal cord are occasionally attached to the pia, but it may be entirely absent (amyelia). In partial rachischisis, the spinal cord is rudimentary in the parts involved. A tumor-like projection, a hernia of

the spinal cord, does not occur in simple rachischisis, but does in that form designated as spina bifida.

By *spina bifida* (cystica) is understood all those cases in which a cystic or a hernia-like tumor projects through a fissure in the vertebral canal.

A frequent consequence of spina bifida is decubitus of the sac and ascending suppurative meningitis.

The condition occurs in varying degrees: (*a*) in the severest forms, namely, those in which careful examination shows there is rachischisis

Fig. 964.

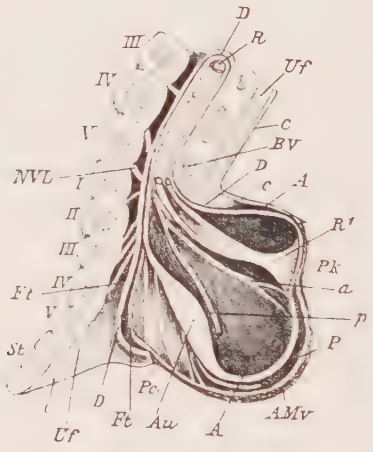


Fig. 965.

Fig. 964.—Lumbar cystic spina bifida with indentation; symmetrical red ulcer-like areas on highest parts of sac. Death of purulent cerebro-spinal meningitis; child 3 weeks old; drawn from fresh specimen; ¼ nat. size.

Fig. 965.—Sacral spina bifida (myelomeningocele); sagittal sect. somewhat to left of median line (redrawn after Recklinghausen). View of r. section surface; R, spinal cord. On the spinal column R-1, rest the two recurrent left IV lumbar nerve roots, their sections visible. The arachnoid A is in the caudal part of the sac detached from the dura and pia, and everted at one place Au, where sacral nerve roots are seen resting on the pia. III, IV, V, the lumbar, and I, II, III, IV, V, the sacral vertebrae; St, the coccyx; a and p, anterior bifurcated, and posterior root of right V lumbar nerve (NVL) passing free through arachnoid sac; AMv, medullo-vascular area; Pk, cranial; Pc, caudal pit; the latter with its canal between dura and arachnoid. Pc is followed by the terminal filament Ft. The spinal column is in contact with Pk; cutis with epidermis; Uf, subcutaneous fatty tissue; BV, lateral part of arch of V lumbar vertebra; nat. size.

with myelo-meningocele, (von Recklinghausen), a tumor appears externally, in the midline of the sacral and lumbar region, more rarely, in the thoracic or cervical regions; it is filled with serum, and projects through the fissure in the vertebral column and through a defect in the dura. The sac, formed principally of the membranes, may be of the size

of an apple to a child's head. The external membrane is usually replaced at the most prominent part of the sac by a richly vascular membrane in the midst of which is a silky red mass—the rudiment of the spinal cord; it appears externally like a granulating wound surface, and is frequently pulled in toward the centre like a funnel. Occasionally the tumor may be divided by a deep median groove, as in a roll (fig. 964). Peripheral to the silky mass is a soft epithelial skin (the zona epithelo-serosa, which corresponds to the pia), then a skin-like zone and finally the normal skin itself. The investigations of von Recklinghausen, confirmed in all particulars by Muscatello, and in almost all by Hildebrand, have shown that the condition is not, as at first thought, a prolapse of the spinal membranes, but is a severe malformation, a rudimentary development of a portion of the back, spinal cord, skin and muscles. (Extensive lit. by Hesse.) A defect in the bodies of the vertebrae prevents union; the dura mater and the external skin are cleft, and thus the medullary depression remains open, and with the pia, lies flat on the dura, and along the edge of the vertebrae. These early conditions might be called simply, "partial rachischisis" (p. 1971). In order to understand the process better, let us imagine that at sometime or other during the course of development we look through a dorsal opening in the outer soft parts through the ossifying vertebral canal and through a defect in the dura, arachnoid and pia, and directly observe the spinal cord which, at this situation, is not enclosed, but denuded, and has become adherent as a silky red mass to the internal surface of the ventral pia seen in the depths of the opening. By lifting up the pia, we see beneath it the subarachnoid meshes, the columns of the spinal cord which are inserted into it, and the nerves passing from it, particularly from the ventral surface of the silky rudiment—through the pulled apart subarachnoid space to and through the dura. To complicate this picture of partial rachischisis, however, there occurs an accumulation of fluid because of increased transudation in the arachnoid space—a hydrops, developed because of chronic inflammation (hydro-meningocele). This lifts the pia mater and the spinal cord from out the edges of the vertebral column, and pushes them outward dorsally as a spherical shaped mass (myelocele). Since the internal surface of the pia is adherent to the cord, this membrane is made to form the outer wall of the sac which is sometimes transparent, or in other cases thickened by fibrous tissue (dura-like). Then as those rudiments of the spinal cord, carried outward through their adherence to the pia advance through the various stages of development, they become changed by the growth of new blood vessels and lie as the silky red mass, upon the pia. This mass, the area medullo-vasculosa (v. Recklinghausen), contains numerous blood vessels, and, among other things, ganglion cells. The area may end in a

caudal and a cranial furrow to which are attached the filum terminale and the spinal cord, which, in these cases, extends far below the position reached by the normal structure; it may be more or less well developed, but is more and more compromised as the sac increases in size through the addition of transudate in the subarachnoid space. Nerves may run freely through the inner part of the sac; they arise in largest part from the area medullo-vasculosa, and in minimum from the part of the spinal cord in the sac and pass into the vertebral canal. The column-like prolongation of the spinal cord may insert in the inside of the sac, and this shows on the surface as an umbilicated depression. In addition to the pia, one of the leaves of the arachnoidea may also help form the sac wall, and then nerves may be seen traversing the wall between two membranes, the outer of which is the pia, the inner, the arachnoidea (fig. 965). To further picture the conditions, we may suppose that we wish to reduce the myelomeningocele. It would be necessary first to remove the arachnoideal fluid, and then to gradually shorten the spinal cord rudiments and the nerves leaving this and the area, until the entire vesicle is turned in, and pulled in and spread out until the internal surface of the pia with the rudiment of the spinal cord lying upon it again takes on its old ventrally situated position in the vertebral furrow as in a partial rachischisis.

E. Neumann described a subcutaneous myelomeningocele as a subtype of the myelomeningocele (aperta) of v. Recklinghausen, and considered it a frequent form of spina bifida; in these cases there is a well developed cutis over the situation of the area medullo-vasculosa. According to Bonsmann (lit.), this subcutaneous myelomeningocele is not altogether rare; it is frequently combined with hydrocephalus internus as is spina bifida in general. This is important because of the external resemblance to (*b*).

(*b*) In a myelocystocele or a hydromyelocele—forms of spina bifida,—the spinal cord projects partially, the tube is closed, but the central canal is dilated. In these cases the dural defect is also present. The wall of the sac is composed of the soft spinal meninges, and, according to von Recklinghausen, may have an area medullo-vasculosa covered by epithelium, and situated internally, usually on the ventral side, and not externally as in a myelomeningocele; the area may, corresponding to the outer wall of the sac, insert the nerve roots. In the cavity itself there are naturally no nerves, and this is an important differential point from *a*. Externally, the skin may be movable over the sac. The chief reason for the occurrence of a myelocystocele lies in an inhibition of the growth in length of the vertebral column, while the spinal cord, which grows normally, is relatively too long and becomes kinked; thus there is a tend-

ency to sacculation where the kinking is most marked, and this pushes outward at the point of least resistance. These weak places may be the result of defects in the vertebral canal (usually lateral) or there may be preexisting openings in the vertebrae or vertebral arches.

(*c*) In a simple meningocele or hydromeningocele, the sac corresponds chiefly to the dorsal side of the dilated subarachnoid space; the spinal cord is not involved.

(*d*) Furthermore, a meningocele from hydrops in the subarachnoid space may be combined with a myelocystocele with a resulting myelocystomeningocele.

In opposition to the opinions described above, other authors believe that the primary trouble resides in a rachischisis of the medullary tube, and they believe the disturbances in other parts such as the bodies of the vertebrae, the muscles and so on, are secondary, *i.e.*, necessary further consequences of the non-union of the medullary tube. The chief foundation for these theories is that the closure of the medullary tube occurs before the formation of the germinal layer for the vertebral column and the skull in the ordinary embryological development. Consult v. Monakow, lit.

See Schwalbe and Gredig, W. R. Thoma, concerning the Chiari-Arnold malformation in spina bifida, *i.e.*, tag-like prolongations of the cerebellum in the vertebral canal and pushing upward of the medulla oblongata. See Henneberg concerning findings in the spinal cord in spina bifida, such as diastomyelia at the transition of the closed spinal cord and the area medullo-vasculosa, the formation of spaces, similar to those of syringomyelia in the closed spinal cord, etc.

Anterior spina bifida is quite unusual (lit. Nieberding, Oehlecker, Budde, Hesse). Of especial interest are the rare cases of spina bifida sacralis anterior, in which the sac of the spina bifida projects into the pelvis like a cystic tumor. This may give rise to gynecologic and diagnostic difficulties. Kroner-Marchand described such a case in which the sac reached the size of a man's head and contained three liters of fluid. In a case described by the author, occurring in a twenty-two year old virgin with a prolapse of the uterus (!), there was a saccular cyst as large as two fists and containing clear fluid; later there appeared symptoms of paralysis; the situation was cleared up at autopsy; there was a large defect in the anterior part of the sacrum where the cyst was inserted; the lower left half of the sacrum and the left side of the coccyx were lacking; death occurred from suppurative meningitis. See also Tilp, Coqui, lit.; in the differential diagnosis, subperitoneal pelvic fibromas and fibromas of the pelvic bones may be considered (Withney, lit., Kuttner). For roentgenological observations, see Altschuh.

Spina bifida occulta, according to roentgenological evidence, is more or less common, and is a sac of a meningocele hidden in the tissues; the sac comes through an opening in the vertebral column or in the sacrum, particularly fissures of the fifth and the fourth or the third sacral vertebrae (Robinson, Graessner); it may have a growth of hair over the skin covering the sac (sacrolumbar hypertrichosis, see Ebstein). Tumor-like new formation of fibrolipomatous and muscular tissue may arise in the sac wall and then spread into the vertebral canal, the dura and the spinal cord (v. Recklinghausen). Borst saw nerve fibres within. A number of the tumors described in the sacral lumbar region as "lipomas," as well as a number of the pseudotails which consist of pendulous caudal lipomas in the sacral region, belong among these pathological changes (lit. Borst, Hesse). These structures are explained thus: through the rupture of a myelomeningocele and the subsequent shrinkage of scars, these tissues are pulled from the depths, since not only the vertebral column but also the dura and even the pia show a defect, and after they are pulled through, they become neoplastic. Frequently there are other, wide spread, disturbances such as club foot, (lit., O. Beck), congenital dislocation of the hip, fissure of the pelvic bones (Chiari), ischias (lit. by Westkott and others). See Hochsinger and p. 1165 concerning spina bifida and congenital fissures of the skull. Fuchs first called attention to the frequent coincidence of occult spina bifida and enuresis nocturna; Hofmann found these conditions combined in 59% of the cases. Jancke showed that there was an inheritable factor through a number of generations. Altschuh believes that occult spina bifida is merely the predisposing cause of the enuresis which is actually caused by other factors (see also Zappert, lit.).

Spina bifida occulta occurs occasionally in prolapse of the uterus; in the prolapse of the new born, it is practically always present (according to Halban and Tandler); the damage to nerve trunks produces an insufficiency and a paralysis of the muscular apparatus of the pelvic floor (Schultz, Burger, Ebeler, Ebeler and Duncker).

Nakayama believes that most sacral tumors are teratomata triphyllica, but this is probably too sweeping a statement. There certainly do occur tumors of simple tissues, as well as the frequent dermoid cysts in the neighborhood of the sacrococcygeal region, which unquestionably are monogerminal, *i.e.*, from snaring off processes between the vertebral canal and the integument; even such a case as that of Kober (lit.) belongs in the same category; it consisted of a gliomatous neuroepithelioma. (See the references of Borst concerning sacral tumors and the literature concerning sacral teratomas by Sklawunos.) See Heusner concerning a cornifying carcinoma in the depths of the sacral region.

The Formation of Cavities in the Spinal Cord (Hydromyelia, Syringomyelia)

Hydromyelia or hydromyelus of the spinal cord is the same as a hydrocephalus internus of the brain. They are occasionally combined. The central canal is dilated, and filled with hydropic fluid. The dilatation may be congenital or acquired, and may be so marked that the spinal cord remains as a relaxed thin-walled sac after the fluid is removed. The dilated central canal, the cross section of which varies in shape—three-cornered, irregular, or rounded,—is lined by cylindrical epithelium and may show a number of out-pouchings, some of them not lined by epithelium (diverticuli); following disturbances in the closure of the medullary furrow, they may be doubled or even tripled.

In many cases nothing certain is known of the development of a diffuse hydromyelia. Attempts have been made to explain the acquired hydromyelia on the basis of inflammatory conditions of the central canal which follow disease foci in the cord itself, and occasionally pus has been found in the central canal (pyomyelia); in other cases, other factors such as lymph stasis obstructing the passage of the cerebrospinal fluid may be mentioned. Among other causes are tumors in the spinal cord or in the skull, crushes and malformations from fractures, previous meningitis, adhesions among the meninges, etc. If the canal is dilated from the presence of blood, or if tubular spaces follow traumatism (rarely spontaneous, lit. Doerr), or if on cross section there are ring shaped or disseminated foci of blood in the spinal cord (Minor), the condition is known as hematomyelia (lit. by Taniguchi). Around the dilated central canal, particularly if acute inflammation is present, but even without this condition, the tissues are softened, whereby the cavity assumes an irregular shape. Occasionally, a more or less vigorous secondary growth of glia is established. Such changes show the close relationship between hydro- and syringomyelia.

In **syringomyelia,** cavities appear in the spinal cord, particularly in the cervical region (cervico-brachial segments), but often, throughout its entire length; in fact, the entire cord occasionally resembles a hollow tube; externally it may be unchanged, or may be swollen so that fluctuation can be observed. The cavity is often so wide that the tip of a finger may easily be placed therein. After removal of the spinal cord and release of a thin watery content, the structure may collapse entirely. When the cord is cut through, it can readily be seen that the internal surfaces of the cavities are covered with tissue masses and sieve-like membranes which insert on the walls. In other cases the spinal cord does not collapse; irregular cavities are seen on cross section, particularly in the region of the posterior commissure and deep in the posterior strands, the latter of which may be markedly separated from each other.

Usually the change is bilateral, but asymmetrical, and more marked on one side than on the other. The cavities have a soft, often villous wall, and contain a thin fluid, or watery, yellow brown, colloid, rarely bloody, content. The cross sections are quite different in the various cavities. Here and there the cavities above are connected with those below by spaces through solid glious masses. Indeed there are cases in which the cross sections show a tissue, soft or firm, often filled with cavities lying in the central parts or in the posterior horns, which tissue may be followed along the entire length of the spinal cord as a cylindrical elongated mass of glia, although there may be no external change in the contour of the cord, or merely a thickening. Alongside of the cavities in the glious part are sometimes soft, white, or gray foci. A lepto- and a pachymeningitis, particularly in the cervical region, are not uncommon. In other cases distinguished by clinical peculiarities, a sharply outlined tumor compresses the spinal cord; secondary cavity formation appears exclusively in the tumor or perhaps also in the remainder of the cord. The anatomical process at the basis of a syringomyelia is thus not uniform.

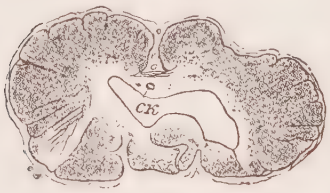


FIG. 966.—Syringomyelia; cross section at level of VIII cervical nerves; CK, central canal; towards the back the cavity surrounded by glia masses (light) (× 3.5). (According to E. Asmus, Uber Syringomyelia. Bibl. med. C. No. 1. 1893) Cassel.

In the various chronic progressive spinal diseases which are catalogued under the name of syringomyelia, there occurs the formation of elongated spaces, which prefer the central parts of the cord; a glia proliferation localizes before, during or after, the cavities are formed, the growth taking place around them or in their neighborhoods in which cavities will eventually form (Schlesinger, lit.). The individual spinal diseases are as follows:

(*a*) Hydromyelia.—See p. 1977. According to Leyden, syringomyelia develops as a result of congenital developmental anomalies in the central canal. A prolongation of the canal persists directed posteriorly, and forms a tubular diverticulum clothed with cylindrical epithelium; this calls forth a secondary overgrowth of glia. Actually, heterotopia of the gray substance in the brain is occasionally seen as an expression of congenital anomalies in the central nervous system, *e.g.*, in a young soldier examined by the author.

(*b*) Furthermore, cavities occur from destruction of tissue in the spinal cord which may be caused by: traumatic degeneration (Schiefferdecker, Schmaus, Wiedemann) particularly when there is hemorrhage, hematomyelia (Minor, Stadelmann, Kolpin), or when a central, so-called tubular hemorrhage, becomes an elongated space in the cord filled with blood

(compare p. 1977). Such a trauma, usually a concussion of the cord, occasionally is the actual cause and not merely predisposing in the development of syringomyelia (Bawli, Schmaus, F. Schultze, Westphal, Nonne, Durck, Siemerling, Fauth, lit.). Petren (lit.) saw hematomyelia follow stretching of the nerves. Birbaum called attention to the possibility of the relation of a birth injury to later syringomyelia (Stoltzenberg). There may also be considered thrombotic or embolic (Chiari) and meningomyelitic processes (Wieting, Saxer, Lasarew), periependymal myelitis (Petren, lit.), or primary myelitic softening (myelite cavitaire). Sadelkow observed tubular hematomyelia in an intramedullary metastasis of carcinoma, and Liebich found the same as a complication of eclampsia. The destruction of tissue may be followed by secondary overgrowth of glia of quite varying degree, but often poorly cellular (sclerosis; Klotz speaks of an irritative hyperplasia). Klotz calls this group (*b*) primary syringomyelia. Petren (lit.) speaks of the sclerotic type of syringomyelia when the replacement of the destroyed parts is accomplished chiefly by sclerotic connective tissue and sclerotic vessels.

(*c*) In a third group there is a primary overgrowth of the glia, richly fibrous or richly cellular, diffuse in the tissues, or circumscribed as a tumor, with or without marked development of the degenerative cavities; in some cases the tissue grows slowly and produces a typical clinical picture (p. 1981); in other cases it shows definite differences. According to Hoffmann, Schlesinger, and others, the majority of cases of typical syringomyelia shows a "primary central gliosis," which produces the above mentioned diffuse glious new growths. This overgrowth may be poorly cellular and tough, (like a sclerosis), or richly cellular, and begins, according to Hoffmann, from the posterior parts of the wall of the central canal, in which germinal cells remain embedded from embryonal life. Attention has also been called to the fact (Stroebe) that the ependymal cells of the central canal and the periependymal prolongations both have the ability to produce glia. (Compare under brain, p. 1920.) Irritation, *e.g.*, a trauma, (as accidental factor, see Durck), leads to the active growth of the tissue. Cavities are frequently formed from the destruction of the newly grown tissue, and these secondary cavities produce the typical picture of syringomyelia (the so-called typical glious or gliomatous syringomyelia), with its preference for the central part (gray substance). But occasionally no larger cavities may be developed. Hydromyelia may be present or absent. It is obvious that these cavities may unite with the central canal by destruction of their walls and therefore appear like diverticuli. Thus cross sections at one level may show this connection and at another level it appears as though there were a simple or congenital reduplication of a closed canal alongside of glious degeneration cavities.

The solid, actual, glioma, circumscribed or diffuse, is, as a rule, solitary; it frequently begins centrally, is usually of more vigorous growth, is more richly cellular and more vascular and can be differentiated, particularly from diffuse gliosis, by its sharper boundaries and by its general character as an ordinary tumor (p. 1933). In gliosis, on the other hand, swelling of the spinal cord is not necessarily coexistent; when, however, the glia overgrowth is more voluminous and more locally distributed, it may nevertheless give the impression of a tumor-like growth. This is spoken of as gliomatosis (Leupold), but there is merely a quantitative and not a qualitative difference between gliosis, gliomatosis, and glioma. The tumor-like glioma may transgress the boundaries of the spinal cord. If it is richly cellular, it may be called a richly cellular glioma, although some call them gliosarcomas (p. 1933). Regressive changes in tumor tissue frequently lead to cavities. According to Hoffmann and others, gliomas must be sharply differentiated from "central gliosis" which produces, as a rule, the clinical picture of syringomyelia, while the glioma grows more rapidly and produces much more noticeable irritative symptoms. It is quite possible to view gliosis as the benign, poorly cellular, variation, which, in its structure, approaches normal glia (or as a hyperplasia), while the glioma is to be regarded as a cellular and richly vascular structure less similar to the glia, producing tumor-like masses (Miura); it must be emphasized, however, that there are transitions between these two forms; this makes quite difficult the differentiation of the kind of a primary new growth in any particular case. Many believe that such a separation into simple and neoplastic glia overgrowth is not possible; thus cases in which a thick layer of glia, or even a tag of glia is formed, are considered by Stroebe, impossible to differentiate from gliomas. Leupold (lit.) believes that gliosis may become a true tumor at any time, as in a case described by him, in which the histological picture was similar to that of an already existing gliosis; gliosis itself is nothing other than an autonomic tumor formation, a glioma.

The case described by Rosenthal as a neuroepithelioma gliomatosis occurring in the thoracic cord is analogous to the tumor of the brain, *i.e.*, of the retina, described on pp. 1925 and 1855; see also the case of Fabritius (neuroepithelioma gliomatosum microcysticum). In the case of Kling, there was central gliosis with cavity formation and a rapidly growing tumor in the lower part of the thoracic cord (neuroepithelioma gliomatosis gangliocellulare). O. Riedel saw a long, hard, pencil-shaped glioma and a soft tumor, entirely separate, of the type of a neuroepithelioma gliomatosum, which caused the spinal cord to be markedly swollen. In the case of Frankel, the tumor showed "sarcomatous" places; see also Thielen.

All causes which lead to the formation of cavities may produce more or less degeneration in the neighboring parts of the spinal cord.

Syringomyelia is a chronic disease. According to Petren, the cases with marked overgrowth of glia die in a few years, while cases running chronic courses, show changes produced by chronic inflammatory processes, (p. 1979), *i.e.*, sclerosis. Because of the typical situation in the cervical cord and spread in the posterior gray matter, the following are the characteristic symptoms: (*a*) progressive muscular atrophy in the upper extremities, almost always bilateral, including the shoulder girdle; (*b*) partial paralysis of sensation, consisting of analgesia and thermic anesthesia in the upper extremities, the head and the neck, while the sense of touch and muscular sensation are little changed; (*c*) vasomotor and trophic disturbances in the skin, the bones, the joints (upper extremities, see p. 1250), bladder, ulcers on the fingers with final gangrene similar to that occurring in Raynaud's disease, but without pain. There then occur mutilation of the phalanges, with frequent spontaneous fractures and luxations. A scoliosis or a kyphoscoliosis is also a frequent complication.

See Sittig (lit.) concerning the combination of multiple sclerosis with syringomyelia. See p. 1850 for syringomyelia and chronic internal hydrocephalus.

In Morvan's disease, or, according to Schlesinger, in Morvan's symptom-complex, known as "paresie analgesique a panaris des extremites superieures," in addition to analgesia and thermic anesthesia, there are tactile anesthesia and trophic disturbances; vesicles and whitlows are particularly apt to occur. This symptom-complex may arise as the result of central diseases (syringomyelia) as well as of peripheral nervous diseases, particularly those of a leprous kind. In fact, Morvan's disease has been looked upon as leprosy (Zambaco), but it is probably not the same (Babes, Lie, Nonne); see also under gangrene in the chapter on skin.

The Histological Changes in Simple Atrophy, Degenerations and Inflammations of the Spinal Cord and Their Consequences

In simple atrophy of the nervous elements of the spinal cord, the nerve fibres and the ganglion cells become smaller, without showing any particular changes in structure; the atrophy increases until there is final disappearance. Atrophy may be a senile change, but may also follow other conditions, *e.g.*, inactivity.

In the course of years, after amputation of an extremity, atrophy may appear in the corresponding side of the spinal cord, the posterior roots, the anterior horns, the columns of Clark, and the pyramidal tracts, which supplied the removed parts with nervous impulses (see p. 1873).

The destruction of parts of the spinal cord as a result of various degenerations due to circulatory disturbances (ischemia, hemorrhages),

toxic or traumatic causes, inflammations of the cord, and the changes in the ganglion cells and nerve fibres are quite similar histologically to those in the brain (see p. 1870 and following). Where the spinal cord substance is destroyed, there are seen the changes (*a*) in the ganglion cells described on p. 1870, and (*b*) in the nerve fibres a destruction of the medullary sheaths and the axis cylinders (p. 1874). The products of destruction are always mixed with fluid, and if much is present, the masses appear like milk of magnesia. This is simple white softening (myelomalacia). If red blood corpuscles escape, there is red softening. If there has been a previous inflammatory process (myelitis), an exudate leaves the blood vessels. The leucocytes often accumulate in the perivascular lymph sheaths, and these inflammatory products are added. The formation of phagocytic fat droplet cells and of pigment granule cells, has been described on p. 1875. (*c*) The vascular interstitial substance may be destroyed along with the nervous elements, or it may be retained. (See p. 1876 concerning these relationships.) Thus, in the first case (*a*), the end result of the reparatory glia overgrowth is a tender and soft, or a tough, harder sclerosis, in which the architecture of the fibres and their direction in the white substance can no longer be recognized. It takes months for the development of an actual sclerosis. Corpora amylacea often appear (p. 1878). If the substance of the spinal cord is focally destroyed, as for instance, after hemorrhage, suppuration, softening, or traumatic degeneration, the most frequent healing result is a connective tissue scar, especially when the pia has also been injured. Smaller foci may be replaced by glia.

Reparatory changes in the ganglion cells, with the exception of the tigroid change (p. 1872), do not occur. Merely minor repairs have been observed in the nerve fibres of humans (Glass), but in animals an axis cylinder stump may grow out from a cell body (Stroebe).

Secondary Degeneration

(Lit. in Appendix)

A nerve fibre is merely a prolongation of a cell which is its trophic center, and is representative of the corresponding ganglion cell; when it is separated from its parent cell body, it degenerates completely throughout its entire length (for more details, see under nerves). If the connections between the cells and the fibres of an entire system are destroyed by a focus of disease, secondary degeneration proceeds along the entire column of nerves. The centripetal or centrifugal direction determines the name ascending or descending degeneration.

Descending degeneration is seen particularly in the pyramidal tracts.

The motor tracts are represented by the lateral and the anterior pyramidal tracts. This path has its (trophic) centre in the motor region

of the cerebrum, in which its ganglion cells are situated; their prolongations are the motor paths which run in the spinal cord in the pyramidal tracts to the anterior horns (the first part of the cortico-muscular tract); in the latter are other motor ganglion cells, and their prolongations form the motor roots and the peripheral motor nerves, which terminate in the end apparatus of the muscles (the second part of the cortico-muscular paths). Compare pages 1889 and 1990.

If the motor path in the brain is interrupted, *e.g.*, in the internal capsule, atrophy overtakes the corresponding pyramidal anterior, and the crossed lateral pyramidal tracts in the spinal cord (fig. 967). If the entire motor path in the spinal cord is broken by trauma or transverse myelitis, the pyramidal tracts undergo bilateral degeneration below the segment (fig. 968-a, b, c). Since the anterior pyramidal tracts extend only as far as the middle or lower thoracic region, they naturally degenerate only when the disease of the spinal cord affects its upper segments.

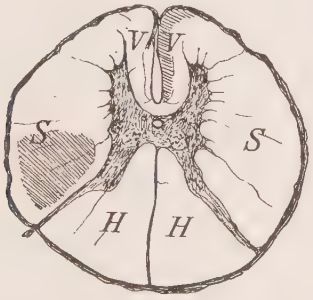


Fig. 967.

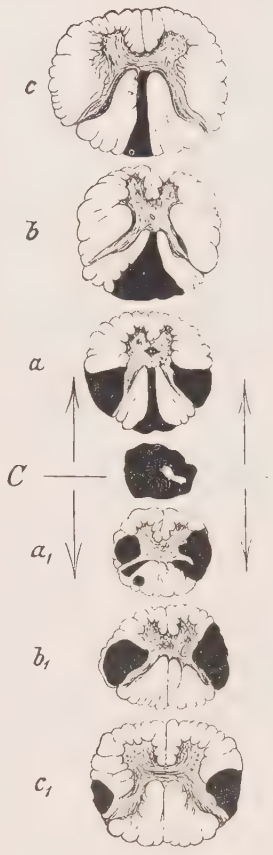


Fig. 968.

Fig. 967.—Cross section through upper thoracic cord. The shaded parts (degenerated) correspond to the right anterior pyramidal tract (direct) and the left lat. pyram. tract (crossed pyramidal tract). The motor conducting path was broken in the right brain. The spinal cord changes are descending degeneration; diagramatic (about × 3.5).

Fig. 968.—Changes in spinal cord due to slow compression from vertebral caries to demonstrate secondary ascending and descending degeneration. (Free after Charcot); a, middle dorsal region; bob, dorsal region; c, cervical expansion; a-1, lower dorsal area; b-1, upper lumbar region; c, lumbar expansion; c, constricted area in lower dorsal cord. The degenerated tracts are shaded black.

If the motor ganglion cells of the anterior horns undergo destruction, the degeneration descends in the second part of the corticomuscular path, *i.e.*, along the motor fibres leaving the spinal cord.

In the posterior strand, four descending degenerated nerve fibre bundles have been observed, among them the comma areas, see p. 1985.

Ascending degeneration. Separation of an ascending sensory nerve fibre from the cell to which it belongs, is followed by destruction of the fibre. Likewise centripetally directed, secondary degenerations of the sensory nerve paths follow primarily degenerated foci in the spinal cord.

The sensory paths are represented in the posterior strand and in the upper thoracic cord, as well as in parts of the lateral strands, *i.e.*, in the cerebellar lateral paths and Gower's bundle. The ascending sensory fibres arise in ganglion cells, which, for the posterior strands, are in the intervertebral ganglia, for the lateral cerebellar strands in Clarke's columns, and for Gower's bundle in the posterior horns. The entire sensory path above the injured place is destroyed. In the posterior paths, however, the degeneration is lost more and more in Burdach's column as it ascends, and in the cervical cord only Goll's column, which is composed of longer fibres, is destroyed (fig. 968-a, b, c). This fact depends upon the peculiar arrangement of the sensory fibres in the posterior columns as described briefly below:

The lowest sensory fibres which enter the posterior strands through the posterior roots lie internally, close to the posterior horns, and course in the lateral parts of the posterior columns in the zone of entrance of the roots, the region of the so-called basal fasciculi of the posterior strands. The new fibres which enter in the next higher zones take a course upward and internally and at the same time slightly obliquely dorsally, force those fibres which have entered first to the centre (medially), into the region of the columns of Goll (Kahler), so that the fibres which lie laterally in the posterior strands in the sacral and lumbar cord, come to lie in a median position in the cervical cord and at the posterior periphery of the transverse section (dorsally), where they form the columns of Goll. Those parts separated by the septum paramedium, the ground bundle of the posterior strands, are called the columns of Burdach (see fig. on p. 1970).

After destruction of the cauda equina, the posterior strands degenerate, following the lesion in the posterior nerve root fibres, and in the lumbar region; this degeneration is almost complete with the exception of a small medial zone and the most anterior parts, while it becomes less and less as the cord is ascended, because above the lesion, newer fibres always enter from intact posterior root fibres, and thus, so to speak, continue to rebuild the columns of Burdach at each level. In the thoracic region, it is finally confined to Goll's columns, so that it may be concluded that these columns are the uninterrupted communications of the fibres from the root zones of the lumbar cord (Singer, Kahler).

In diseases affecting the cord transversely, such as compression, section, etc., the descending degeneration occurs chiefly in the pyramidal tracts and the ascending degeneration in the posterior columns, the cere-

bello-lateral tracts, and in the bundles of Gower (see fig. 968). (Consult the anatomical remarks concerning the sensory neurone on p. 1998.)

Two small comma areas in the posterior strands also degenerate after transverse lesions in the cervical or the upper thoracic cord (F. Schultze); these lie on either side in the region of the posterior horns (Matuszewski, lit.).

The Histological Changes in Secondary Degeneration.—These secondary degenerations appear soon after interruption of communication; they are always simultaneous in the entire region of the involved nerve fibres. (In primary column sclerosis, fibre after fibre becomes involved in the process.) Even two days following an injury, the destruction appears in the medullary sheath (Stroebe); a few days after this, isolated axis cylinders begin to undergo degeneration. A few months after, the degenerated cords are soft, cloudy and white. The meshes of the glia which is preserved, contain fluid, or degeneration masses, or fat droplet cells. The spaces of the glia gradually become obliterated, and granule cells remain for long lengths of time in the meshes (months, years). Isolated granule cells may also be found in the perivascular lymph sheaths and in the bordering pia. Corpora amylacea are often present in large numbers. The perivascular connective tissue thickens, and the degenerated column shrinks more and more and finally comes to consist of a gray, fairly tough tissue (secondary gliosis or sclerosis).

Brown-Sequard's paraplegia is a particular symptom complex occurring in (experimental) unilateral section, or in pathological breaks in the continuity from tumors, caries, syphilitic processes, etc. Paralysis develops with hyperesthesia and increase in temperature (vasomotor paralysis) amounting to five tenths to one degree C. on the corresponding side; there is anesthesia on the opposite side. The half lesion is found most frequently in the dorsal cord. If the condition follows traumatism, the prognosis is relatively favorable.

The Focal or Diffuse Inflammatory and Degenerative Processes Known as Myelitis

By "myelitis" is understood focal, or perhaps also diffuse inflammatory and degenerative or softening processes in the spinal cord, which involve the white substance (leucomyelitis), or the gray substance (poliomyelitis), or both. There may be differentiated: (*a*) true myelitis (acute infiltrative myelitis, see Schmitt, lit.). It must be agreed that this is inflammatory in origin as may be deduced from its etiology, in which various infectious diseases play the principal part, and from its histological criteria, *i.e.*, hyperemia, inflammatory edema, exudation, emigration of lymphocytes or leucocytes, swelling and degeneration of the nervous parenchyma with the

appearance of granule or phagocytic cells (see p. 1875), from the marked collection of cells in the neighborhood of the vessels and in their walls, as well as in the interstitial tissue, which form focal cellular infiltrates composed of varying kinds of cells coming by emigration and by proliferation (see p. 1875), and finally, further proof of its inflammatory nature is given by the demonstration, even though difficult, of the actual causes of the infection. If these latter are not found, it may be assumed that there is a bacterial or toxic influence, especially because in experimental bacterial myelitis, the bacilli, as a rule, soon disappear. The inflammations arise from hematogenous or lymphogenous routes or by direct extension.

Among the infectious diseases may be mentioned: the acute exanthemata such as variola, scarlet fever, measles, whooping cough, typhoid fever, influenza (Spiegel), erysipelas, pneumonia, localized suppurative foci such as a paranephric abscess (H. Boehm), a streptococcus abscess of the prostate (Gross), a staphylococcus panaritium; even in gonorrhea, syphilis and tuberculosis, a non-specific form of myelitis may be called forth by the respective organisms or toxins. Malaria may also be mentioned. The changes in the spinal cord are a myelitis, and frequently a meningomyelitis. Experimentally, myelitis has been produced in many general infections by various bacteria such as the pyogenic cocci, colon bacilli, typhoid bacilli, diphtheria bacilli, as well as by merely bacterial toxins. Actual inflammation of the cord by extension may come from inflammatory processes in the vertebrae, as in tuberculous caries, from the dura, even from the skull cavity in suppurative meningitis, or from the meninges as in cerebrospinal meningitis, because infections follow the blood vessels to the spinal cord, but often only to the border or outer zone (meningomyelitis).

But by the term "myelitis" is also understood, (*b*) such changes which begin as a degeneration, a myelo degeneration, or as a hemorrhage, or as nutritional disturbances from intoxications, traumata (concussions) and others. In their further course, they usually show softening (myelomalacia), which may finally produce pictures similar both macroscopically and microscopically to changes brought about primarily by inflammation. It is consequently not always possible to differentiate between inflammatory and simple degenerative changes just as in neuritis (see Schmaus); when it is possible, however, it is usually best to make the separation.

Thus, for example, even a simple white softening of the spinal cord, a myelomalacia, following crushing traumata, has been called a compression myelitis (p. 2007); the softening which follows ligation of the abdominal aorta has also been called a myelitis, in spite of the fact that it is not a primary inflammatory process, but is much more of primary degenerative nature, although it is very rapidly followed by reactive growth of the glia

and cellular infiltration. As a result, the histological pictures of primary degeneration and primary inflammation have the following chief points in common: destruction of nerve fibres, appearance of overgrowth of glia, and wandering cells. On this basis then, the condition is spoken of as myelitis. The substances undergoing destruction are the inflammatory irritants.

Among the agents causing toxemic myelo degeneration (the so-called hematogenous acute myelitis) may be mentioned: carbon monoxide, chloroform, bisulphide of carbon, the poisonous substances which arise during the course of chronic tuberculosis, tumors, particularly carcinoma (see Nonne, also p. 2004) diabetes, syphilis and the above mentioned acute infectious diseases. The botulinus toxin of food poisoning (p. 762) may also be mentioned (Hinze, lit.), as well as intravenously injected salvarsan, which may produce, in addition, a hemorrhagic encephalitis (Schmorl, Chiari, Socin, lit.).

The details of the histological changes in myelitis are those which are commonly seen in inflammatory and simple degenerative destruction, and have been described on pp. 1981 and 1982.

The course may be acute or chronic. In acute myelitis, the destroyed tissue becomes soft and cannot be cut smoothly. On cross section, the most characteristic appearance is displayed by the white matter (leucomyelitis), the architecture of which is destroyed, and from which a soft, gray yellow, or gray white, or red speckled white substance bulges forth as a pulp. In a smear preparation, granule cells may be found quite readily. The name "hemorrhagic myelitis" is used when considerable hemorrhage is present; the end result is red softening.

FIG. 969.—White substance of spinal cord, normal. Cross section of medullated nerves; sheaths dark; axis cylinders unstained. (Med. power.)

Hemorrhages into the substance of the spinal cord have been observed in some of the cases of infectious diseases as mentioned on p. 1986 as well as in epidemic cerebrospinal meningitis (Ebmeyer, Dessauer, lit.).

There is also a suppurative form which leads to spinal cord abscesses. These are quite rare in comparison to the incidence of brain abscesses. The pyogenic organisms may enter from direct trauma, or via the lymphatics or blood vessels. In the latter case, infectious emboli have been carried from a primary focus present elsewhere in the body; thus, *e.g.*, a thrombophlebitis, an endocarditis, or a focus in the lungs, etc. In bronchiectasis, Chiari (lit.) saw metastatic myelitis with numerous elongated foci of pus (Silvast). Abscesses may also occur in gonorrhea (Wolff). In rare instances, a tubular-shaped abscess may stretch throughout the entire length of the spinal cord.

In the experiments by Hoche and Marinesco, the introduction of infectious organisms into the cord brought about a myelitis only when there was coincident disturbance in the circulation (lycopodium injected into the vessels); for further literature, see Schmaus.

In chronic myelitis, the diseased places occasionally become tougher in consistency and appear gray and glassy since the destruction of the nervous parts is accompanied, or followed by a vigorous growth of glia (gray degeneration, sclerosis).

Gray degeneration is also the ordinary end result of healing of myelitis, if the individual is not too old, and if the serious destruction of the glia tissues by the inflammatory process is not too extensive, (*e.g.*, when there are abscesses).

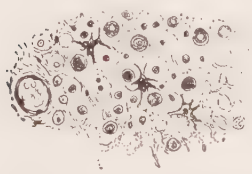


Fig. 970.—Gray degeneration of white substance, atrophy of nerve fibres. The fibrous glia tissue with blood vessels and so-called spider cells is very prominent; gaps in the glia tissue due to disappearance of nerves; occasional granule cells in the gaps; some dark laminated corpora amylacea scattered here and there. (Medium power.)

Hematogenous myelitis is the form which the clinician calls myelitis, and which he separates quite sharply from the columnar and systemic diseases as well as from poliomyelitis which is particularly common in children. Myelitis is an acute or subacute condition, and rarely chronic.

Infections as mentioned on p. 1986 are the chief etiological agents. But intoxications must also be considered (p. 1986). A myelitis with a particularly bad prognosis occurs during pregnancy and in the puerperium; infectious organisms are supposed to be the cause, but others believe it may be an autointoxication (v. Hosslin, lit., Rosenberger and Schmincke). The course varies; those following the acute infectious diseases usually have the best prognosis. The insidious progressive varieties, and those which lead to a transverse myelitis, usually do not heal. According to its extent, there may be differentiated: general myelitis where the process spreads over huge areas of the spinal cord; central myelitis, in which the central gray part is particularly involved; the inflammatory invaders may perhaps have spread inside of the central canal; disseminated myelitis, in which numerous inflammatory foci are formed in various parts of the cord; and transverse myelitis, where the entire cross section is diseased.

The clinical symptoms of transverse myelitis are comparable to those following transverse sections of the spinal cord, and are somewhat as follows: in dorsal myelitis, with its situation in the thoracic cord: paraplegia of the lower extremities without degenerative atrophy, with stiffness and increase of the tendon reflexes. Anesthesia; the sensitivity is diminished or entirely lost in the legs and the gluteal region; finally, there are girdle pains. Reflex irritability in the legs is retained, often increased. There is bladder and rectal paralysis. Decubitus.

In lumbar myelitis: paraplegia of the legs, but relaxed, degenerative paralysis. The tendon reflexes disappear, the skin reflexes are retained. The bladder and rectum are paralyzed.

In cervical myelitis situated in the lower part of the cervical cord: atrophic paralysis of the arms, spastic paralysis of the legs, anesthesia of both arms and legs and the gluteal region, narrowing of the pupils, disturbances of respiration, particularly of inspiration because of involvement of the abdominal and intercostal muscles; otherwise the symptoms are like those of dorsal myelitis. When the region involved is in the upper part of the cervical cord, the paralyses involve the diaphragm, and finally there are bulbar symptoms.

See p. 1993 concerning Landry's paralysis.

Multiple Sclerosis

See under brain, p. 1911, for the part played by the spinal cord in multiple or disseminated sclerosis, sclerosis cerebrospinalis multiplex.

Primary Column and System Diseases of the Spinal Cord

Diseases Involving the Motor Neurones.—Nissl in particular, has opposed the teaching concerning neurones (Die Neuronenlehre und ihre Jena 1903). According to this teaching, the individual neurones have merely contact communications among themselves. The investigations of Apathy and Bethe, O. Schultze and others, however, have fairly well established the theory that there is no such anatomic nor physiologic independence of the neurones, but that the nervous system throughout its entire extent is one continuous whole. This, according to Bethe, is accomplished by skeins and networks of neurofibrils, which are inside of the dendrites and neurones, and pass through the ganglion cells, and surround these structures with a reticular system, the so-called "Golgi's nets." The fibrils are the conductive elements of the nerves. We, however, believe it is useful, from theoretical as well as didactic grounds, (see also van Gehuchten im Handb. d. path. Anat. des Nervensystems, von Flatau unsw. Berlin 1903) to imagine that the neurone is a functional nervous individual, even though not anatomically, nor genetically so. For a good review, see Gierlich and Herxheimer.

Thus we speak of motor as well as of sensory neurones; by neurones (Ramon y Cajal, Waldeyer), we understand an anatomic and physiologic unit composed of three parts, a ganglion cell (nerve cell), with its afferent protoplasmic prolongation (dendrite) and its efferent axis cylinder or neurite (nerve prolongation). Both the neurites and the dendrites end in fine branching structures. In the motor neurones, some of the nerve cells lie in the cortex of the brain, some in the anterior horns of the spinal

cord and some in the bulbar nuclei, while the nerve prolongations lie in the pyramidal tracts, the anterior roots and the motor peripheral nerves.

From the motor neurones, there is formed the cortico-muscular communicating tract. This leads from both sides of the brain from the cortical motor centres (psychomotor centres of the cerebral cortex) through the pyramidal tracts to the anterior horns. Here a fibre comes into communication with one of the ganglion cells in the anterior horn. The sum of these neurones forms the first part of the motor path. The dendrites of this motor ganglion cell in the anterior horn then form a second neurone with a corresponding cell in the motor peripheral part entering and leading to the muscle. These neurones form the second part of the cortico-muscular communicating tract.

Acute anterior poliomyelitis, Heine-Medin's disease. This acute atrophic spinal paralysis, or acute poliomyelitis of the anterior horns, occurs most frequently in childhood, occasionally as a concomitant of infectious diseases such as scarlet fever, whooping cough and measles; it is produced by an infectious agent, traveling in the blood vessels or lymphatic systems of the spinal cord. It usually appears suddenly with high fever, which, as a rule, may last but a few hours or days, is occasionally epidemic (Geirsvold), and leads quickly, often over night, to relaxed paralysis of one or more members—usually first of the left leg,—and later to atrophy with resultant contraction and deformities of the paralyzed parts.

Children of any age, from sucklings to puberty, may be attacked; adults are rarely its victims.

The paralyses reach their highest degree at one stroke; all following changes lead to improvements. They are transitory in largest part, but finally concentrate themselves on isolated muscles, *e.g.*, in the leg. The rapidly ensuing degenerative atrophy of the muscles may be masked by fat and connective tissue overgrowth. See also under paralytic pes equinus and calcaneus on p. 1270, figs. I and II.

Newer investigations have shown that this disease, the infectivity of which was first shown by Wickman in 1905, is possibly due to a filtrable, living, invisible virus (Flexner and Lewis, Landsteiner and Levaditi). Its infectivity for monkeys was first shown by Landsteiner and Popper, and confirmed by the above mentioned authors, and by Knopfelmacher, Leiner and v. Wiesner, Roemer, Kling-Wernstedt-Petterson, and others (see lit. in appendix). Flexner and Noguchi have grown the organism anerobically as a very small "globoid body" which stains with Gram and Giemsa stains, is about 0.15 to 0.3 microns in diameter, passes through a Berkefeld filter, after which it appears in pairs or in chains. It has been demonstrated in the brain and spinal cord, in the nasal passages, in the saliva, in the lumbar

puncture fluid, in the blood, in the lymph nodes, the spleen and the liver; these findings correspond to the pathologic anatomic and clinical picture of the disease in humans. As routes of infection in the experiments, there have been tried direct intracranial inoculation of material from the spinal cord, intraneural inoculation into the peripheral nerves, introduction into the peritoneal cavity, the blood stream, the subcutaneous tissues, the anterior chamber of the eye, the peripheral lymph nodes and particularly the naso-pharynx.

Carriers of the Infectious Agent.—Natural means of transmission. The excretions, particularly those of the nose, contain and carry the infectious organism (see Flexner). It is possible to infect monkeys by inoculations of the nasal washings (Flexner-Clark-Fraser). Natural transmission occurs, just as in other infectious diseases, through direct or indirect contact from person to person, according to Flexner, from the nasal and pharyngeal mucous membranes to similar mucous membranes (according to Lugard, from mouth to mouth). Healthy persons who have been in contact with poliomyelitis cases may become intermediate carriers of the infection. Flies may also be implicated, at least as mechanical carriers of the virus (Flexner).

As the investigations of Wickman, Harbitz and Scheel and others have shown, the disease involves the entire spinal cord including the lepto-meninx, as a diffuse infiltrating inflammation, especially marked around the vessels, and most markedly involving the smaller parts of the anterior horns which are the situations particularly rich in vessels. The same changes may be found also in the medulla and in larger parts of the brain itself. While Harbitz and Scheel believe that the meninges are always the site of first localization (meningomyelitis), from which structures the inflammation (round cell infiltration) spreads along the perivascular lymph sheaths of the pia to the central nervous substance, Wickman emphasizes that the virus, on the contrary, reaches the meninges through the lymph vessels from the nervous substance, and then settles in the anterior horns. There appear several clinical types according to whether the inflammation localizes in one or another segment, and there are, furthermore, transitional forms to acute bulbar paralysis, to meningoencephalitis, or even to an entire transverse myelitis. As a rule, the intervertebral ganglia also show cellular infiltrates and degenerative changes (Flexner and Lewis, lit., Landsteiner and Levaditi, lit.). In this stage of the process the peripheral nerves show no changes.

In the earlier stages there is an acute interstitial and infiltrating non-suppurative inflammation which is predominantly and narrowly localized to the vascular apparatus. These foci of infiltration are characterized by marked richness of cells, particularly those of the lymphocytic variety

(according to Marburg, almost exclusively large mononuclear cells, but Wickman calls them polyblasts or large lymphocytes), but there are also a number of leucocytes (Harbitz and Scheel); as Hauptli (lit.) showed with the oxidase reaction (method of Graff, p. 196), during the first few days, they are present in large numbers in the tissue infiltration as an important component for the removal of debris. Then there occurs destruction of the ganglion cells with cloudiness, swelling, fatty degeneration, infiltration with round cells (neuronophages, see Wickman); these cells always lie in the middle of inflammatory foci, but these foci may still contain intact ganglion cells, and may also be present outside of the gray matter. The earlier observations of the early stages (see, for example, Matthes and Praetorius, lit.), tell of the changes in the gray substance of the anterior horns and lay great stress upon these findings. In addition to these, there are found edema, hemorrhages, stretching and thrombosis of the vessels (according to Wickman, these are artefacts following formaldehyde fixation), lymphocytes, plasma cells and granule cells in the perivascular sheaths. Later investigations emphasize the involvement of the glia in the following stages; this proliferation is probably a reparatory process and removes damaged nervous tissue by wandering glial cells; it finally overbalances the actual inflammatory infiltrations (Hauptli). Whereas Wickman, Harbitz and Scheel and a large number of other experimenters (see Wickman and lit. on p. 1990) never found bacteria, even in early foci (those diplococci found in the spinal fluid by F. Schultze, Geirsvold and others are probably contaminating organisms because of their size), nevertheless, Flexner and Noguchi were able to find the globoid bodies in the diseased tissues in humans and in the experimental animals. (Lit. to p. 1990.)

Attention has been called on pp. 1901 and 1994 to the similarity of the anatomic changes of rabies and poliomyelitis; the former is, in fact, an acute poliomyelitis caused by a filtrable, invisible, living virus which retains its properties for a long time (many years), after drying and conservation in glycerin and in frozen preparations; furthermore, it, like poliomyelitis, spreads along the nerves to the spinal cord after peripheral application of the virus (see lit. to p. 1990). Hauptli believes the histological changes in poliomyelitis are quite similar to those in epidemic lethargic encephalitis, and cerebral localizations of the inflammatory processes can always be found in fatal cases of poliomyelitis with the difference that the destruction of the ganglion cells is always more extensive in the spinal cord (Wegelin). The same remarks hold true for Borna's disease of the horse, a non-bacterial parasitic affection caused by the invasion of specific inclusions in the ganglion cells similar to the Negri bodies in rabies; the same is also true for the organisms of chicken cholera and distemper, as a

product of the reaction of the ganglion cells to the invasion of an organized parasitic agent (Joest, lit.). See also Tobler, Techendorf (lit.).

If the disease is studied in later stages (after months or years), there is found a lack of ganglion cells and nerve fibres in more or less extensive areas of the cord corresponding to the regions with permanent paralyses. This is mostly in the anterior horn of one side in a segment of the spinal cord, which in the cervical or the lumbosacral cord, becomes several millimeters to centimeters thick; it may be recognized macroscopically by the diminution in size of the corresponding half of the spinal cord when the white substance also participates.

But a decrease in the number of ganglion cells may sometimes be demonstrated outside of the actual region of permanent paralysis. As a rule, corresponding to the disappearance of the ganglion cells, the nerve fibres in the anterior roots are atrophic. Fibres in the gray substance may also disappear in part. Occasionally, degenerative changes take place in the posterior horns as well as in the anterior lateral strands. Secondary atrophy develops in the corresponding spinal roots, nerves and muscles. See p. 1271 concerning paralytic contractures. Lateral pseudohernias of the abdomen are described on p. 736.

Subacute and chronic anterior poliomyelitis usually occur in older life. Microscopically, the lesion is chiefly a disappearance of the ganglion cells and nerves in the anterior horns. The atrophic paralyses prefer whole complexes of muscle (atrophy en masse), particularly those of the legs, the upper arm and shoulder girdle. The muscles are relaxed, the tendon reflexes absent.

Landry's paralysis, or acute ascending paralysis may be mentioned in passing. This disease is caused by various toxic or infectious processes, and produces a very characteristic symptom-complex; in some cases there are no positive anatomic findings, but in most cases there are changes in the spinal cord in the form of true myelitic processes, particularly in the gray substance (central myelitis).

The disease may be presented as follows: after mild prodromes (feeling of illness) there soon develops a flaccid paralysis of the legs progressing to completion. The tendon and skin reflexes are diminished or absent; the electrical response of the paralyzed muscles and their sensitivity are, in general, not altered. In the course of a few days, the paralysis progresses to the musculature of the back and the arms. This is followed by involvement of the muscles of deglutition, articulation and respiration; bulbar symptoms appear, and in eight to ten days, seldom earlier or later (after weeks), death occurs from asphyxia. According to Oppenheim, the characteristic of these cases is the rapid sequence of flaccid paralysis starting in the legs and passing into the gluteal regions

and the arms to the bulbar nerves, but in many other details the case histories are variable. In some cases healing takes place, inasmuch as the paralysis slowly disappears.

The anatomic findings are not consistent. Occasionally, it is necessary to search for finer changes in the ganglion cells. In some cases inflammatory vascular changes, particularly as an interstitial inflammatory reaction, have been found; in others there is parenchymatous swelling. Disseminated foci have been observed in the medulla oblongata and in the spinal cord. According to Wickman, these changes are similar in all respects to those of poliomyelitis (p. 1992). Marburg also emphasizes this close relationship; the disease as an infectious entity usually involves the entire nervous system; according to its localization it calls forth a poliomyelitis, an encephalitis, Landry's paralysis, a neuritis, or a herpes zoster. Remlinger described a case of rabies which was masked under the picture of Landry's paralysis, an observation of interest in connection with Wickman's description of the similarity of the pathological process in poliomyelitis and rabies (see pp. 1901 and 1992). Many authors believe that neuritis is the foundation of the disease. At autopsy, there have been repeatedly seen swelling of the spleen, and of the mesenteric nodes, and hemorrhagic infarcts in the lungs and intestines.

Etiologically, infectious and toxic products are held responsible. Thus the disease has been thought to follow variola, diphtheria, typhoid fever, influenza, pneumonia, septic diseases in general, and even anthrax, and the causes of these infections have ofttimes been demonstrated. Alcoholism and lues (Hempel) have also been mentioned as possible causes. According to the very plausible opinion of Oppenheim, it seems probable that the poison affects the motor tracts in the spinal cord, the medulla oblongata and the peripheral nerves; it may produce paralysis without extensive anatomical changes (pure functional disturbance), but it occasionally does produce severe anatomic damage; on the whole, however, the damage to the trophic centers and the tracts is not very marked, since there is no disappearance of musculature. Experiments with animals in which bacteria and toxins have been used illustrate the many aspects of these myelitic symptoms (see Schmaus for lit. concerning Landry's paralysis).

Amyotrophic Lateral Sclerosis (Charcot). Its anatomical foundation is a combination of atrophy of the pyramidal tracts, the anterior horns, the anterior roots and the motor nerves of the spinal cord. The same changes are found in the medulla oblongata, in the motor nerve nuclei of the hypoglossal, facial, vagus and accessory, and the motor trigeminal nerves; the symptoms are those of a progressive bulbar paralysis. If the degeneration extends further upward into the pons, the cerebral peduncles,

the internal capsules, the cerebrum, and to the pyramidal cells of the paracentral lobe or the central convolution, which occasionally occurs, the entire corticomuscular transmission path is diseased, *i.e.*, both motor neurones (see p. 1983).

The muscles show a degenerative atrophy as in spinal muscular atrophy. The tendon phenomena are markedly increased. The symptoms of typical cases are composed of three isolated complexes, namely, those of an anterior poliomyelitis, of a spastic spinal paralysis, and of bulbar paralysis.

The disease is seen chiefly in middle life. Its etiology is unknown; traumata (Giese, Erb and others, see Marburg), congenital anlage (Strumpell), initiated through accident, (Klieneberger), cold, over-exertion in the sense of over-fatigue (Edinger), fright (Oppenheim), vascular toxemic (Haenel) causes, have all been postulated. The usual course lasts from two to four years, occasionally much longer, even up to twenty-five years. Death occurs most often from aspiration pneumonia.

FIG. 971.—Amyotrophic lateral sclerosis; a, b, c, cervical, thoracic, lumbar spinal cord. The diseased parts shaded black are the lateral pyramidal tracts (diagram after Charcot)

Progressive bulbar paralysis (paralysis labio-glosso-laryngea progressiva) may appear independently without amyotrophic lateral sclerosis, and depends upon a progressive disappearance of the motor nerve nuclei in the medulla oblongata and in the pons. The progressive atrophy is particularly prone to occur in the nuclei of the hypoglossal and facial nerves, the motor part of the glossopharyngeo-vago-accessorius, and occasionally in the motor trigeminal also.

The results are paralysis and change in the muscles of the lips, the tongue, the gums, the pharynx, the larynx, and the muscles of mastication. Disturbances of speech in the form of dysarthria are usually the first symptoms, followed by difficulties in swallowing, phonation and respiration. In the spinal cord, the characteristic changes of amyotrophic lateral sclerosis and progressive muscular atrophy are often found; they may be combined, either one or the other being first in the order of occurrence.

The causes are shrouded in darkness (syphilis ?). Death usually occurs in one to three years from inanition, bronchitis, or aspiration pneumonia.

The so-called myasthenic paralysis (or myasthenia gravis pseudo-paralytica, Oppenheim) is a bulbar paralysis without anatomic findings. In addition to this symptom, the fatigue (myasthenia), ptosis, weakness of the trunk, back, and leg muscles are particularly marked. Younger individuals are preferred. Death often occurs suddenly from asphyxia,

occasionally also from aspiration pneumonia following relaxation of the muscles of deglutition. The course is of a varying, occasionally long, duration. Healing is problematic. The muscles do not change in volume nor in irritability. Sensory and sensible changes are usually lacking. The anatomic findings in the nervous apparatus are negative, even if the disease has persisted for years. On the other hand, certain changes have been found in the muscles, which have been described as lymphoid cellular infiltrates, or as "light staining" muscle cells (Knoblauch), or as a degenerative myositis or as a hyalin change. Very little is definitely known concerning their causes. Oppenheim (lit.) believes there is probably a congenital anlage at fault. It is also thought that toxic products of tumors are implicated since mediastinal tumors (see p. 504), have occasionally been observed coincidently. According to Chvostek, there is possibly a functional disturbance in the parathyroids (p. 542). Hart calls attention to the relationship with the thymus, which, as the beneficial influence of extirpation of a hyperplastic thymus showed, may have something to do with the myasthenia; it is also said that the intramuscular cellular collections of lymphocytic character are possibly due to a specific effect of the thymus; (see Markeloff, lit., Tobias, Stern, Lewandowky). Mathias (lit.) proposes the name "myopathia alba lymphoides" (asthenica).

In the **spinal form of progressive muscular atrophy** (amyotrophia spinalis progressiva—typus Duchenne-Aran), the chief findings in the cord are changes in the gray anterior horns, which consist of atrophy of the motor ganglion cells, the nerve fibers, the anterior roots and the muscle nerves; the muscles themselves participate in the atrophy. Therefore the entire second part of the cortico-muscular communicating tracts, the peripheral neurone, is diseased (see pp. 1983 and 1991).

As a rule, the disease begins insidiously in middle life and usually attacks the cervical region first. As a rule also, the atrophy is accompanied by fibrillary tremors beginning in the small muscles of both hands; it produces a deepening of the interosseous spaces, a flattening of the balls of the thumb and little finger, a claw hand, monkey hand, etc.; it spreads very slowly, but jumps to isolated muscles causing atrophy until, during the course of more or less years, it involves most of the arm, shoulder, and back musculature. In other cases the atrophy begins in the shoulder and back musculature. Finally, the muscles of respiration, particularly the diaphragm, are attacked, or symptoms of progressive bulbar paralysis are added which then lead to death. The atrophy is the primary symptom and the paralysis is a consequence. The tendon phenomena in the arms are diminished. The paralyzed muscles are pale red, red yellow or pure yellow and are marbled with yellow stripes of fat. The fibers become more and more diminished in size, their contents undergo granular and fatty

degeneration; there finally remains merely the tube of sarcolemma with the muscle nucleus. The pectoralis may, for example, be changed into an almost transparent, white striped membrane. Pseudo-hypertrophy does not develop.

That form of varying degrees of muscle atrophy called myotonia congenita (Oppenheim), is caused in the majority of cases by a disease of the motor ganglion cells in the spinal cord, and is said by Neumann, (lit.) to be a fetal form of the infantile and the progressive muscle atrophy of adults. (Lit. by Marburg, Thorspecken, Cassirer, Kaumheimer.)

Primary sclerosis of the lateral tracts, and especially primary symmetrical sclerosis of the lateral pyramidal tracts is the cause of so-called spastic spinal paralysis, or lateral sclerosis (according to Erb, Charcot and Strumpell). This well defined clinical condition consists of spastic paralysis without atrophy of the muscles, and of increase in the tendon reflexes.

It usually appears between the twentieth and fortieth years of life and is practically always chronic. Disturbances in gait and in rigidity of the muscles are the first symptoms, as a rule. Later, the legs become contracted in the extended position so that the patient pushes himself forward with small steps; pain is absent. There is also a form which seems to be hereditary or familial, particularly in male members of a family (Strumpell, Dobrochotow, lit., Reitter, Bremer; see also Merzbacher, laws of inheritance in relation to these familial hereditary diseases). The etiology is known only in part (lues, trauma, intoxication, as with lead, acute infectious diseases, puerperium).

Anatomically pure forms are only isolated. Usually the autopsy discloses some other spinal disease such as multiple sclerosis, chronic myelitis, combined column sclerosis, hydromyelia, tumors, etc. Primary isolated lateral sclerosis is often found anatomically in paralytic dementia. (Lit. by Erb, Redlich, Strumpell.)

Congenital or early appearing spastic paraparesis (the congenital spastic joint condition, or Little's disease), is probably not a purely spinal but also a cerebral, *i.e.*, a cerebrospinal, disease (cerebral paralysis of children). Difficult and protracted labor with traumatism from forceps and consequent intracranial hemorrhages and their consequences, porencephalia, etc. (see p. 1810), wherein the pyramidal tracts are most frequently involved, are given as the chief causes. But failure of development in the motor region may also be responsible in certain cases. Not rarely, other developmental anomalies are present in addition (lit. by Newmark and others).

Diseases Involving the Sensory Neurones.—In the sensory neurones, the ganglion cells are present in the spinal ganglia (intervertebral ganglia).

They possess one nerve prolongation which quickly divides into fibers passing peripherally and centripetally; the former is a sensory fiber of the peripheral nerve; the latter enters the spinal cord through a posterior root, where it divides in the posterior column into a chief branch passing upward to the medulla oblongata, and into a smaller descending branch; the latter is soon lost in the gray substance. From the stem as well as from the longitudinal branches of the posterior root fibers, there pass collaterals into the gray substance at all heights, but they are particularly numerous in the middle third of Burdach's column and in the zone of entrance of the roots; they then split up toward both the sensory and the motor ganglion cells and thus complete communications with these two (p. 1990), which is important for the reflexes. Some of the posterior root fibers remain in the posterior columns, and enter the medulla oblongata into the so-called posterior column nuclei. In the thoracic cord, a large number of the posterior root fibers enter Clark's column.

The course of the sensory fibers toward the medulla oblongata and the brain: the columns of Goll and Burdach traverse the medulla oblongata to the nucleus gracilis and nucleus caudatus, groups of ganglion cells which lie at the upper end of the posterior columns. Neurites of these ganglion cells pass upward, cross the midline and join with the already crossed sensory tracts of the spinal cord. Then the combined sensory path passes through the medulla oblongata and the pons; whereupon the fibers divide into (*a*) those which pass through the tegmental region into the posterior fossa until they reach the internal capsule, where they lie dorsal to the motor pyramidal tracts, and then pass through the corona radiata into the region of the cortex of the parietal lobes; (*b*) those going to the lenticular nucleus; and (*c*) those which reach the corpora quadrigemina.

Tabes Dorsalis.—The most striking and important of the pathologic anatomic changes in tabes are the progressive disappearances of nerves in the posterior columns and the gray degeneration (sclerosis) which this brings about. In outspoken cases, the posterior columns can be seen to be smaller, even macroscopically; they do not appear swollen, but sunken in, are hardened, and following the disappearance of the nerve substance are no longer cloudy white, but transparent and gray. Actually, the entire spinal cord is small and thin in serious cases. The pia is thickened on the posterior side of the cord, dull, and often adherent.

More careful examination, however, reveals that there is an atrophy of the gray substance, particularly in the posterior horns, in which Lissauer's bundles are the chief participants, but there is also a disappearance of the fibers of Clark's columns which come from the posterior roots. (Lissauer's columns are the bundles which come from the posterior roots after their entrance into the spinal cord, and which lie on the periphery of

the posterior horns). As a rule, the posterior roots also undergo atrophy and may appear gray. It has been believed that the point of origin of the process is in this situation (Leyden, Schaffer and others), inasmuch as the degeneration of the posterior column is an intramedullary progression of the process in the roots. In many cases atrophy can be demonstrated in the spinal ganglia, involving the fibers and cells. It also seems that there is usually an atrophy of the peripheral sensory nerves.

The type of changes in the roots is still subject to controversy. Obersteiner and Redlich point to a compression from meningeal or pial contracting processes. Since the posterior roots are not uniformly involved by this meningitis, nor equally, and since certain roots may even remain uninvolved, thus producing segmentary and also bilateral differences, it appears to Obersteiner not unlikely that tabes cannot be regarded as a system disease in the strict sense of the word. Schaffer assumes that infiltrative and vascular meningeal processes are quite important in the disease of the roots. Dejerine also believes that there is an early inflammation of the posterior roots which results in tabes. Richter, who considers tabes a truly syphilitic disease, with its chief site localized in the subarachnoid space, believes that the tabetic changes originate as a granulation tissue brought forth by the spirochetes (few in number); this granulation tissue appears in the root nerve (radicular nerve, shortly before the union of both roots and toward the spinal side of the ganglion), and invades the posterior roots (mechanical and toxic damage). The changes in the spinal ganglia are said to be secondary to the above process; the degeneration in the posterior column may be explained by the degeneration in the posterior roots. There then follows an unsystematic, ascending secondary degeneration. This of course leaves open the question of the character of the system degeneration, since the damage to the fibers in the posterior strands can be found to be elective in most tabetics; perhaps the two processes go together. Jakob believes there is a local activity of spirochetes with a resulting connective tissue reaction in the extramedullary parts of the roots which leads to the gradually developing posterior fiber degeneration; on the other hand, another, hitherto undiscovered toxin may be the cause.

While Richter considers diffuse meningitis of the pia, *i.e.*, the cellular infiltrate of this membrane, a syphilitic, but an inconstant process, which has nothing to do with the course of the tabetic process but is merely an accompanying symptom, Thorne believes that pial and intramedullary perivascular plasma cell infiltrations are equally important and of the same significance as the corresponding infiltrates in the brain in ordinary paralysis.

Others are inclined to believe that the center of the entire tabetic process is disease of the spinal ganglia, or they are at least influenced by the noxa producing tabes. (see P. Marie, early opinion, Oppenheim); this is then followed by secondary degeneration in Clark's columns, the posterior roots and the peripheral nerves. Occasionally, degenerative changes can actually be found in the spinal ganglion cells (Wollenberg, Oppenheim, Stroebe and others). But these changes, as Obersteiner emphasizes, are only of mild degree. In view of the above mentioned ideas, they are merely secondary.

FIG. 972. FIGS. 973 to 975.

FIG. 972.—Tabes dorsalis; cervical cord, advanced case. The posterior columns are almost completely degenerated (light), especially the columns of Goll which are separated by a septum from the columns of Burdach. Weigert stain. (Slides stained with Weigert's hematoxylin stain or with Pal's modification of it show the medullary sheaths black blue; areas which lost their sheaths appear light.)

FIGS. 973 to 975.—Tabes dorsalis. (Black) degeneration of posterior columns; a, cervical cord, degeneration of Goll's columns; b, thoracic cord lateral root zones degenerated; c, lumbar cord shows complete degeneration of posterior columns (diagram).

According to some authors, even the beginning of the tabetic process is in the peripheral nerves, not excluding their peripheral end apparatuses.

The changes in the nerve fibers and columns consist of a very slowly progressing destruction of fibers with the appearance of very few granule cells. Even in advanced cases, granule cells, perivascular cell collections, and preserved nerve fibers are met in the gray foci between atrophic fibers and naked axis cylinders; finally the nervous elements disappear entirely, and there remains a finely fibrillar, thick, rich, overgrowth of glia (sclerosis), perhaps containing many corpora amylacea (fig. 970, p. 1989).

For the catabolic processes in tabes, see details by Faworsky.

The beginning of the disease in the columns is usually in the upper lumbar cord in Burdach's column in two symmetrical fields near the entrance of the roots. Later, the posterior columns in the lumbar and

thoracic cord may be totally degenerated to very small fields near the posterior commissure. In the cervical cord, the subsequent secondary ascending degeneration appears more and more in the columns of Goll, and may, in the beginning at least, be confined to this structure, whereas in later stages, the other external parts of the posterior column are also diseased. Only small parts remain entirely free (fig. 972). The process may, in rare cases, begin in the cervical cord. Upward, the disease occasionally reaches the medulla oblongata; the spinal roots of the trigeminal and the solitary bundles may atrophy, as well as the nuclei of the muscles of the eye ball. But the cerebral nerves themselves may be directly damaged by a true syphilitic process situated chiefly in the intra-arachnoid spaces, as seen particularly in the nervus radicularis (Richter; p. 1999). The optic nerves are frequently diseased. According to Palich-Szanto, the process begins in the intracranial portion or in the chiasm; exudative processes follow direct invasion of spirochetes (Stargardt); further, according to Richter, the exudate is characterized by pial plasma cell infiltrates. This inflammatory process leads to secondary atrophy of the medullated nerve fibers, while the neuroglia grows; the nerves become gray and atrophic. Gray foci also appear occasionally in the brain itself.

Etiology of Tabes.—It can no longer be doubted that syphilis is an important factor, as assumed for many years (by Fournier and Erb in opposing the opinion of Leyden); but even though it is a syphilitic disease, it is influenced very little by antisyphilitic medication, a fact which is not surprising, considering the anatomic changes in outspoken tabes. Although Strumpell and Gowers pointed out that in no case of the disease could syphilis be demonstrated beyond doubt, yet the positive Wassermann reactions of the blood serum of sixty to seventy per cent of the cases (Nonne and Holzmann), together with an increase in the globulin content and the number of cells in the cerebrospinal fluid, and finally a positive Wassermann in the fluid, all lead to its classification with great probability as a syphilitic disease. (See the significance of examinations of the spinal fluid from this viewpoint by Schonfeld, extensive lit.) To this may be added the fact that Noguchi found the spirochetes in tabes, even though, at first, only in a few cases. But Nonne (lit.) will not characterize paralysis and tabes as a spirillosis of the brain and spinal cord; Erb calls tabes a chronic form of late syphilis, while Eichhorst and Strumpell speak of a post syphilitic condition. The investigations of Richter, Jakob, and others show the specific character of the initial histological changes which are much more important than the consecutive degenerative changes presented by the completed anatomic picture which have nothing in common with the pathologic anatomic changes in other visceral syphilis. But there do occur cases, even though rarely, of combinations of tabes

with specific syphilitic processes such as gummata and vascular lesions (see p. 1916). (See Fieandt; for others, see lit. to p. 2003.)

All other factors formerly considered etiologically important such as overexertion, fright, alcoholism, sexual excesses, cold, are believed to be merely predisposing (Erb), or to accentuate the changes; the relation of tabes to trauma must be considered in the latter sense (E. Schultze). According to Edinger's overexertion or exhaustion theory, degeneration

FIG. 976.—Pellagra. Sharp limitation of the pigmentation just above the hands. Man 35 years old. L. H.

of the cord would follow insufficient nutrition in the presence of functionally increased use just as in animals which have previously been made anemic, and then are forced to undertake marked bodily exertion; in these latter, there occurred degenerations in the posterior strands; in an organism infected by syphilis and poisoned by its toxins, the nutritional conditions are hampered, so that the equilibrium between nerve fibers and interstitial substance is disturbed, and the latter gains the upper hand. The experiences of Düring in Anatolia, in which country there is said to be much syphilis but very little tabes (also in other countries

such as Japan, Abyssinia and others), do not fit in with the syphilitic etiology of this disease, but his statements are doubted by others. Perhaps there is an as yet unknown factor which helps syphilis produce tabes; that syphilis is an initiative cause cannot be doubted. Tuczek showed that chronic ergot poisoning may be symptomatically and anatomically allied to tabes, but is not progressive.

Pellagra is a disease which occurs, among other places, in upper Italy, south of France, Spain and Rumania; for a long time it was believed that the etiology was dietary (Tizzoni, Nageotte and Riche, lit., and Camurri); there are combined system lesions in this disease, which particularly involve the columns of Goll in addition to the anterior lateral strands (Leyden); poisonous bodies which develop in decomposed maize (for more details, see Babes and Sion), have been held responsible for them (Tuczek), but others dispute this view (lit. by Alessandrini and Scala); it is now believed that the diseae is an avitaminosis (see Stepp, Bickel), due to a qualitatively insufficient diet with too little vitamins (Rondoni, Perroncito, Babes, see also lit. by Kozowski, Raubitschek).

Translator's Remarks.—Pellagra apparently is seen more frequently in recent years; the translator encountered twelve cases in the last two years. At autopsy of two cases, men 21 and 32 years old, atrophy of the entire walls of greater part of the small intestines was found with the signs of an acute enteritis in one. No other definite lesions were discovered except for the pigmentation and scaliness of the upper extremities distal to the middle of the forearms. For discussion of the disease, lit., treatment, etc., see Goldberger J., Medicine 5: 79: 1926; DeLamar Lect. J. Hopk. Univ. School Hygiene 1927, page 128. For pellagra in Philadelphia, see Knowles F. C. and Ludy J. B., M. Clin. N. Amer. 10: 431: 1926.

Strumpell assumed that the anatomic changes in tabes were brought about by chemical poisons which might be syphilitic toxins electively choosing certain specialized parts of the nervous apparatus. It is much more frequent in men than in women (see K. Mendel and Tobias, lit.). It usually begins between the twentieth and fiftieth years (5, 10, 15–20 years after the syphilitic infection, rarely earlier or later). It also occurs in individuals with congenital lues, more frequently in females (Baumgart); v. Hosslin described tabes in a 52 year old woman on the basis of a congenital lues since she had never had sexual intercourse, and her hymen was intact. Not rarely, tabes is combined with progressive paralysis. Contact infection from a tabetic (or a paralytic) is unknown (Kaufmann-Wolf).

Of the many symptoms which appear in this chronic disease lasting one to two decades or even three or more, there may be mentioned:

1. **In the Early Stages.** (*a*) Loss of knee jerks, (*b*) reflex fixation of the pupil (Robertson); early myosis; (*c*) lancinating pains; (*d*) mild sensory disturbances in the lower extremities; (*e*) temporary paralysis of muscles of the eye; (double vision), etc. This stage lasts months (two to twenty), or perhaps years (interesting cases cited by Oppenheim and Malaise); according to Westphal, such exceptional stationary cases must be regarded as being practically healed.

2. **Stage of Complete Development.** (*a*) Development of the ataxia in which the motor power is retained, but in which there is a characteristic disturbance in gait; (*b*) the Romberg sign (unsteadiness with closed eyelids); (*c*) parathesias of various kinds; (*d*) disturbances of bladder function; (*e*) crises, particularly gastric, with attacks of vomiting and pain which are observed in a small minority of the cases, but which appear in these even in the earliest stages of the disease; (*f*) sexual power early diminished, finally, almost entirely extinguished (impotence).

3. **The End Stage.** Complications from the bladder, the kidneys (cysto-pyelitis) lead to death. There are atrophy of the muscles, and, even though infrequently, actual paralysis of the legs; this may be spoken of as the paralytic stage. For osteo-arthropathy, see p. 1250, and consult Jansen. Lit. in appendix.

Combined System Diseases

In rare instances there is a combination of degeneration from the very beginning in the sensory fibers of the posterior strands and degeneration of the pyramidal tracts or the cerebello-lateral tracts. Usually the combination is that of posterior column and lateral column disease.

According to Lewandowsky, they are usually unsystematic, extraordinarily extensive, mildly myelitic conditions (funicular myelitis, see also p. 1988). They occur particularly in pernicious anemia (see lit. p. 191, consult Thoenes) and leukemia, but also in the cachexias of carcinoma and other such diseases (lit. by Henneberg).

Clinically, interesting combinations correspond to destruction of various neurone groups. The pictures become more complicated when the symptoms of isolated posterior column disease (tabes-atony of the muscles, ataxia), and the symptoms of isolated lateral strand disease (spastic spinal paralysis—increase of muscle tonus, increase of the tendon phenomena, motor weakness) are combined to various degrees. According to whether the predominant lesions are in the posterior or lateral strands (Westphal), there appear combined symptom-complexes, of which, according to Oppenheim, two deserve the diagnosis of combined system disease:

1. the symptom-complex of "spastic spinal paralysis:" ataxia, lancinating pains, weakness of the bladder, fixation of pupils and other tabetic symptoms; 2. the symptom-complex of tabes—with which motor weakness is combined or even preceded.

Friedreich's disease, or hereditary ataxia, a very rare, usually familial disease, in which brothers and sisters are attacked, and which appears first in childhood and is very chronic, belongs to the combined system diseases of the posterior and lateral columns; Goll's columns degenerate in toto, Burdach's columns almost entirely, along with the cerebello lateral strands, the pyramidal lateral tracts, and Clark's columns, in which the fibers disappear and the ganglion cells are destroyed. Gower's strands are usually involved to some degree. On the other hand, atrophy of the posterior roots and the peripheral nerves is not constant and not marked. Many believe that there is congenital atrophy of the cerebellum and that this is the foundation of the disease. Among the clinical symptoms in addition to the ataxia, are observed: scoliosis, loss of tendon reflexes, choreiform movements, nystagmus, disturbances in speech; in the differentiation from tabes, the most important tabetic symptoms such as visceral crises, atrophy of the optic nerves, reflex pupil disturbances, paralyses of the eye muscles are lacking; bladder and rectal troubles are very infrequent. Nevertheless, many, as for example, Leube, believe that Friedreich's ataxia is merely a modification of tabes. (Lit. by Oppenheim.)

Traumatic Injuries to the Spinal Cord

Acute traumatic lesions of the spinal cord follow incisions, stab wounds, and gun shot wounds. Degeneration appears not only in the neighborhood of the actual injury, but also spreads to a fairly wide zone; there are swelling and necrosis of the nerve fibers. Regeneration of the nervous tissue does not take place (Yamada, lit.). Following this, there occur the secondary degenerations in the columns (p. 1984). The situation of the wound finally becomes a glia-connective tissue scar, and the atrophic spinal cord is contracted. They occur through partial injuries to the vertebrae consisting of contusions and fractures, and if splintering breaks from gun shot wounds are excluded, they are usually compression fractures, with loosening of fragments, dislocations, distortions, and complete luxations of the lateral joints of the vertebrae (rotation luxation); also of total injury or destruction of the vertebral column which, according to Kocher, must be differentiated into total luxation, a dislocation of the vertebrae produced by bilateral luxation of the lateral joints, with destruction and pushing apart of the ligaments, and total luxation fractures. In this case, the lateral joints are luxated and the body of the vertebra is fractured at the same time.

Kocher differentiates two types: a compression-luxation-fracture, and a luxation-oblique fracture; in the latter, the break runs obliquely from behind forward, and there follows a particularly marked shoving apart, with crushing of the spinal cord. Pure luxation without accompanying injury to the bone is rare (Borchard). See Quetsch for accidental injuries to the vertebral column.

Furthermore, fractures and luxations with angular deformities (kyphosis) and lateral displacement are frequent in vertebrae pathological from tuberculosis or carcinoma.

Fig. 977.

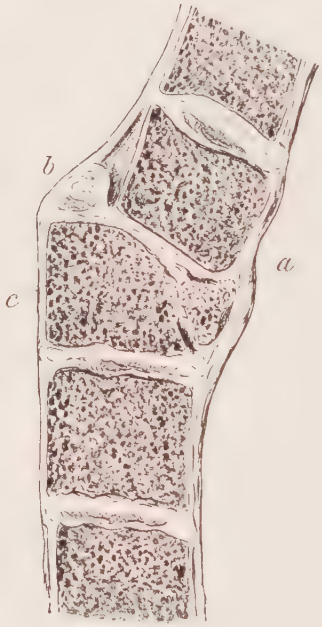


Fig. 978.

Fig. 977.—Compression fracture with wedge-shaped malformation of 11th thoracic vertebra; 5 months before fall on feet from scaffolding. The broken posterior fragments healed but compressed cord, myelomalacia; cystitis, renal abscesses; Mason 37 years old; $\frac{9}{10}$ nat. size.

Fig. 978.—Consolidated fracture of VII cervical vertebra (c) with breaking off of an anterior wedge (a) and luxation of spine. Compression myelitis of the whole transverse section. A smaller fragment at b. Fall from a wagon, 4½ months before; $\frac{9}{10}$ nat. size.

Luxation is the unilateral (abduction luxation) or total separation of the surfaces of the joints. The upper vertebra which is called the luxated, is usually pushed forward. By diastasis is understood a dislocation of the vertebrae in the longitudinal direction. The spinal cord may be directly crushed in any of these conditions; thus, for example, in fractures of the vertebral bodies, the intervertebral discs move closer, and the crushed, flattened, softened spongiosa of the spinal cord is compromised, or more frequently, the vertebral body is pushed forward and the vertebral column

is kinked with the angle open anteriorly. The healthy body of the upper vertebra is pushed forward, while the crushed, wedge-shaped body of the lower is pushed backward, and the spinal cord is compressed against the vertebral arch (fig. 977). In other cases, a loose piece of bone is forced into the vertebral canal and thus compromises the cord, but the dura, as a rule, is not torn. Usually the fragments are from the bodies, but, for example, in the cervical vertebrae, they may come from the arch, or a spinous process.

The spinal cord is torn relatively often by fragments of bone; total cross section is rare, but may be observed from marked concussion or stretching without fracture. Very frequently, traumatic fractures and luxations are accompanied by hemorrhages between the periosteum and the dura, usually from tears in the venous plexuses, and these in their turn compress the spinal cord. A hematomyelia may also follow traumata to the vertebral column (pp. 1977 and 1978); it may occur with an intact vertebral column.

The production of such columnar hemorrhages is particularly easy in the gray substance, especially that of the posterior horns of the dorsal cord. This has been substantiated experimentally (Goldscheider and Flatau, see also Luxenburger). Concussion may produce transient or severe lasting damage to the nerve cells (lit. Arndt). See also Oppenheim, Winkler and Jochmann, concerning traumatic spinal diseases (hematomyelia, myelorrhexis), with intact vertebral column. For the medico-legal aspect, see Deetz.

For comprehensive reviews of traumata to the vertebral column and their consequences, see de Quervain and Payr. See Quetsch, concerning injuries of the vertebral column by accidents; for gun shot wounds, see Lemberg.

The nervous elements are destroyed at the injured places; finally the glia and the connective tissue also. There then appears a simple white, or, in hemorrhage, a red softening. The tissues become pulpy, almost fluid, and meshy. The meshwork is a remnant of the septa. The neighborhood is yellow, infiltrated by round and granule cells. Disturbances of the blood and lymph circulation lead to white and red softening in situations distant from the injured part. Fat granule cells or pigment granule cells may remain for long times in the meshy tissue, whereby the situation, which becomes more and more flattened (fig. 959, p. 1963), is occasionally yellow or brown in color. The final occurrence is sclerosis or connective tissue formation, the latter particularly in places which become infected, or are intensively destroyed or crushed; thus, more often in total luxations and fractures.

See Spatz, concerning degenerative and regenerative changes in the nerves and glia following experimental spinal cord injuries.

Supervening inflammation can be recognized microscopically by the criteria discussed on p. 1986; a wide border zone may be saturated with edema or exudate. Occasionally an abscess is developed, followed perhaps by meningitis.

See the remarks on p. 1986 concerning myelitis.

FIG. 979.—Spontaneous compression fracture of carcinomatous II thoracic vertebra (a) with resulting kyphosis and transverse lesion. Total transverse paralysis developed in 8 weeks. Osteoplastic carcinoma nodule in luxated I thoracic vertebra (b); woman 55 years old; 5 years before I. breast removed for solid scirrhous carcinoma (locally healed); 9/10 nat. size.

Usually the entire cross section is involved, and a picture appears as in acute transverse myelitis (p. 1988). There is interruption of all the paths of the spinal cord, and the spinal centers, *i.e.*, the ganglion cells, disappear, while the peripheral motor nerves undergo degeneration. As a rule, the outcome is fatal. Paralysis of the bladder is followed by infectious cystitis and pyelonephritis, with frequent formation of concrements in the pelves of the kidneys (Kurt Muller, Seefisch, lit.).

Chronic traumatic injury to the spinal cord occurs from slowly progressing compression (the so-called compression myelitis). The spinal cord undergoes degeneration (myelo-malacia, p. 1988). Clinically, it is shown by paralysis (pressure paralysis). Various conditions interfere with the space in the vertebral canal, as follows:

(*a*) **Tuberculous caries of the vertebral column;** these are not the cases in which there is a sudden breaking down of the vertebral column with marked compression from the dislocated vertebrae, but the gradual compression caused most frequently by an extra-dural collection of pus, forcing the dura more and more against the spinal cord, to which may be added the pressure produced by a slowly developing kyphosis of the vertebral column at this situation. Rarely, there are flattening and spindle-shaped thinning of the spinal cord. Non-tuberculous kypho-scoliosis seldom produces compression. (*b*) **Secondary tumors of the dura,** particularly carcinomas, which follow carcinomatous infiltration of the bodies of the vertebrae; occasionally a thick layer of tumor tissue surrounds both the spinal cord and the spinal roots. (*c*) **Carcinomatous** or other neoplastic vertebral bodies gradually break down with dislocation, so that a compression myelitis is produced (fig. 979). (*d*) More rarely, compression is caused by a primary tumor of the dura (fig. 959, p. 1963) or of the soft mem-

branes of the spinal cord (fig. 961). (*e*) By extravasations of blood around the spinal cord or in the central canal. (*f*) By pachymeningitis, particularly the syphilitic variety, as well as (*g*) by parasites, particularly echinococci. The syndrome of compression may also occur in intramedullary processes, *e.g.*, carcinomatous transverse myelodegeneration (p. 1986).

Individual spinal cords vary in their susceptibility. Occasionally, the symptoms of myelodegeneration appear suddenly, sometimes very late, and sometimes when but a small amount of space is compromised. Softening of the cord occurs by (1) direct pressure; the spinal cord appears flat and displaced, is usually soft, more rarely sclerotic. But often a myelomalacia is produced (2) by compression acting secondarily through disturbance of the circulation of the blood, the lymph (Fickler), or the cerebrospinal fluid; this leads to edema of the nervous tissue so that the latter gradually undergoes destruction by swelling and softening. A reactive inflammation may appear as a secondary process (Bornstein). The entire cross section may be involved in a transverse myelomalacia. This is, of course, followed by secondary degeneration of the columns (p. 1982). It is the white paths which first degenerate, while the ganglion cells retain their integrity for longer lengths of time. According to the degree of compression, in time, there appears a sclerosis or connective tissue overgrowth in the region.

Repair of the traumatized spinal cord is possible only in such cases in which the tissues have been damaged to a minor degree by concussion, or when the edema of the nervous tissue disappears without having produced degenerative destruction. See Wiedemann (lit.) concerning traumatic degeneration of the spinal cord. According to Fickler and Schmaus, the "compression myelitis" caused by tuberculous caries of the vertebrae, is usually a stasis as well as an inflammatory edema which swells the nervous parts, and which later leads to destruction of the nervous elements (white softening) after the appearance of marked exudation and emigration of cells, or as a consequence of the pachymeningitis which accompanies the caries. This explains the fact that the spinal cord is not compressed nor reduced in size, but on the contrary, is usually swollen in the situation where there is believed to be narrowing of the vertebral canal and at which, in vivo, the site of compression is thought to be.

As an appendix, the so-called **caisson disease** may be mentioned. This is observed in divers and workers in caissons in the building of bridges, who work under a pressure of three to four atmospheres; a shorter or longer time after sudden exit into the ordinary air they show severe symptoms such as pain in the muscles and joints of the legs, cerebral and spinal paralyses, which, in a few minutes, may become actual paraplegias.

When the transition from compression to the ordinary atmosphere is sudden, there may also be hemorrhages from the nose, the ear, the bronchi and into the joint cavities. Death has occurred from apoplexy, either immediately, or some hours, or days (1 to 20) later, with disturbances of the heart and lungs. According to the experiments of Heller, Mager and v. Schrotter (lit.), there is a relief of gas following the sudden transition from high to ordinary atmospheric pressures; the gas is almost exclusively nitrogen, which, under the higher pressure is dissolved in the blood in saturated solution, and absorbed by the tissues (v. Leyden); under ordinary atmospheric pressure, it diffuses back into the capillary blood; the bubbles of gas become free in the blood stream and lead to gas embolism (Hoche). Thus the smaller vessels of the spinal cord, particularly in the lateral or posterior columns of the cervical and lower thoracic regions, as well as larger cerebral vessels may be obstructed. Ischemic softened foci and tears of vessels and surrounding nerve fibers may occur; at least, this was found in animals after rapid decompression from high (10 to 18 atmospheres) pressure (v. Leyden, Nikiforoff). The further course leads to sclerosis, the formation of cavities, and secondary degenerations. The cardiac and pulmonary symptoms (pulmonary hemorrhages) are also due to gas embolism. R. Heller (Zanger), lit.; see also Bornstein.

Infectious Granulation Tumors of the Spinal Cord

Tuberculosis.—It has been mentioned that a tuberculous meningitis may be followed by tuberculous myelitis by progression of the granulation tissue along the vessels; this causes degeneration of the interstitial tissue with swelling and softening. Larger conglomerate, or the so-called solitary tubercles, often beginning as isolated nodules in the gray substance, are rare; they are rounded, hard, yellow green, perhaps concentrically laminated (Obolinsky); central necrosis occurs finally, but they are seldom cavitated, while they are often surrounded by zones of softening. Even when they have reached considerable size (hazel nuts), they may be surrounded by a thin layer of spinal cord, and thus not be visible externally; nevertheless the cord may show a spindle-shaped swelling and the membranes be secondarily involved. According to their situation (most commonly in the lumbar cord, see Mohr), and their size, there will occur interruptions in communication and subsequent secondary degeneration. But even in the middle of a tubercle, intact naked axis cylinders are sometimes found (L. Muller). Origin: Some are hematogenous, metastatic from the lungs or bones or the joints; others are lymphogenous from vertebral caries or spinal meningeal tuberculosis (lit. by Doerr). Very rarely, an independent disseminated tuberculosis is found in the substance of the spinal cord without involvement of the meninges; this leads to myelitic

degenerative foci of varying extent, and is followed by secondary column degeneration (Raymond's nodular tuberculosis); see v. Rencz.

Syphilis.—Gummas in preferred situations in the substance of the spinal cord, are rare; they are relatively often multiple, and are usually combined with similar growths in the meninges. For the relationship of tabes to syphilis, see p. 2001 and following. Rarely, there occur severe syphilitic vessel changes, among them endarteritis obliterans, of quite local distribution. These lead to chronic focal degeneration of the cord with varying clinical pictures, but they may, after a long duration, lead acutely to softening (myelomalacia), and to sudden paraplegia (Staemmler, lit.). In other cases, paraplegia is caused by syphilitic meningitis in the cervical region, the growth of which closely surrounds the spinal cord. See Nageotte and Riche concerning spinal cord syphilis.

Leprosy.—In isolated cases there have been demonstrated degenerations of the nerve fibers and ganglion cells (Tschirjew), inflammatory foci, hemorrhages, lepra bacilli in the interstitial substance of the spinal cord (Chassiotis) as well as in the ganglion cells of the spinal ganglia (Sudakewitch). See Lie concerning degeneration of the posterior strands. Literature by Stahlberg.

Primary Tumors of the Spinal Cord

There occur gliomas, gliomyxomas, the so-called gliosarcomas (richly cellular gliomas), spindle cell sarcomas, myxosarcomas, hemangiomas (very rare) (Pinner, Roman, lit.), cavernous-like structures (O. Meyer and Kohler), etc. Epithelial tumors which may come from the epithelium of the central canal are very unusual (lit. by Bruns, Schlesinger, Borst).

In a rare case observed by Frick, a teratoma of the vertebral canal, after separation of the spinal cord, pushed two lobes into the canal.

All tumors with the exception of gliomas are very seldom seen. Gliomas, as a rule, arise in the gray substance (in the cervical region), and form elongated, central masses, without sharp boundaries; they usually do not involve the membranes, but may spread diffusely through them (lit. Rutimeyer). If a central glioma undergoes destruction, particularly in a thinner part, and becomes filled with cavities, the condition is reckoned along with syringomyelia (p. 1979). In many cases the development of the glioma can be traced back to the embryonal period (p. 1930); their growth is very slow. Traumata are said to predispose to the formation of tumors (p. 1930). Very infrequently the melanotic sarcoma is seen, as described by Pick; he believes it could be traced to the displacement of chromatophores from the subectodermal tissue during the development of the spinal cord. Neurinomas (nerve cell tumors, see p. 2029), characterized by uniform tender fibrillar structure, yellow

staining with van Gieson method, transverse position of the nuclei, appear as tumors of the roots (Rheinberger and lit. by Antoni). Other so-called "true neuromas" of the spinal cord which have been described, have been later recognized as artefacts (van Gieson, Hanau), and are composed of normal white substance which has oozed out through tears in the pia produced unintentionally during removal of the spinal cord. The same is true of the "innumerable glious tumors" of Freudweiler, as Fr. Schultze showed in a frozen and thawed spinal cord. Even "heterotopia of the gray substance" (see p. 1861 for lit.), may be an artefact. In true heterotopias all gross deformities of the cord are missing.

By spinal cord tumors, the clinician means not only true tumors, but also other structures which produce the same sort of local effect on the spinal cord such as cystic serous meningitis, tuberculosis, gumma, parasites, either of the spinal cord itself or of the membranes, etc., (xanthochromia of the cerebro-spinal fluid, see p. 1847).

True metastatic tumors, sarcomas and carcinomas, are quite rare (the case of Taniguchi was an intramedullary carcinoma nodule following bronchial carcinoma; for more details see Siefert, Gallavardin and Varay and lit. by Miller); Belz described the metastasis of a Grawitz tumor (p. 1395) in the lumbar cord. Sadelkow saw a tubular spinal cord hemorrhage follow an intramedullary carcinoma metastasis. It is more frequent for tumors of the meninges to produce isolated nodules in the substance of the spinal cord. Naturally a metastatic carcinoma of the vertebral column may spread to the cord. Less often a tumor of the pleura extends by way of the intercostal nerves to the spinal pia (see Heyde and Curschmann).

Parasites

Cysticerci have been found in the substance of the spinal cord, but echinococci occur only outside; very rarely they appear primarily in the meninges and usually from the vertebral column of the upper thoracic region; they have been described, however, in the lumbar and sacral vertebrae, and in the vertebral canal; they are mostly extradural. In the vertebral canal they appear from subpleural or pelvic connective tissues. Death may occur from total compression of the spinal cord (lit. by Borchardt and Rothmann, Ciuffini, Boge).

Peripheral Nerves

Anatomical Remarks.—The peripheral nervous system is composed of peripheral nerves, ganglia, and nerve endings.

The peripheral nerves are composed of medullated nerve fibers. (Non-medullated fibers comprise the chief fiber mass of the sympathetic

nervous system.) The medullated nerve fiber is composed of (1) the axis cylinder, 1 to 10 microns in thickness; it is finely striped longitudinally, corresponding to its composition of fibrillae (neurofibrillae), which are bound together by neuroplasma, the mass of which varies according to the thickness of the nerve fibers, from 10 to 50μ (see Sobotta's atlas). (2) The medullary sheath is composed of myelin and surrounds the axis cylinder. It is segmented (Ranvier's segments), a segment comprising the parts between two indentations. The myelin sheath is not composed of an excretion from the nerve cell protoplasm, as many assume, but is a derivative of Schwann's cells (Broman). (3) The sheath of Schwann or the neurilemma, is a fine membrane, in the segments of which are nuclei surrounded by some protoplasm, which are also called the cells of Schwann's sheath, Schwann's cells, or peripheral glial cells. The neurilemma is not a connective tissue but an ectodermal product and comes from particular cells, the "lemnoblasts" of glious origin. A connective tissue covering, the so-called Henle's or endoneural sheath, surrounds the isolated nerve fibers. Groups of medullated nerve fibers (nerve bundles) are surrounded by a connective tissue sheath built up in concentric layers (perineurium). Between the separate nerve bundles in a larger nerve, there appears fine, fat-containing connective tissue which surrounds the entire nerve and is called the epineurium.

Nerve Degeneration, and Nerve Inflammation

When a nerve disappears without inflammatory reaction, *e.g.*, a nerve fiber disappearing after destruction of its corresponding ganglion cells, the condition is spoken of as simple degeneration or atrophy; this process attacks chiefly the parenchyma of the nerve, *i.e.*, the medullary sheath and the axis cylinder. If the process is accompanied by exudation, cellular infiltration and tissue growth in the perineurium or in the interstitial tissue, and if the nerve parenchyma is destroyed, or if degenerative changes in the parenchyma are complicated by inflammatory changes in the connective tissue, the condition is spoken of as neuritis, and there are differentiated perineuritis, interstitial, and parenchymatous neuritis, even though the separation cannot always be made sharply.

Whereas a perineuritis and an interstitial neuritis in their acute forms are characterized by hyperemia, exudation, emigration of leucocytes and later by inflammatory connective tissue production, as a true inflammatory process, these changes are masked in parenchymatous neuritis by the simple degeneration, so that a strict division is ofttimes impossible; many authors consider a simple degeneration a neuritis, and call the condition parenchymatous neuritis in contrast to true (interstitial) neuritis.

There may also be differentiated: primary and secondary degeneration of nerve fibers; by the former is meant a process brought about, not by destruction of the trophic center or by separation of the communication between the center and the part, but by direct action on the nerve fiber itself.

Simple nerve degeneration or atrophy is seen when a nerve is sectioned (traumatic degeneration). (For gunshot wounds of the nerves, see Spielmeyer and Berblinger, lit.) The entire peripheral part of the nerve including its end apparatus in the muscle and the muscle itself, undergoes secondary degeneration following section of the communication between the nerve fiber and its corresponding cell, as stated in the so-called law of Waller, while the fiber of the central stump does not remain entirely unchanged, but shows much milder lesions which may be overlooked for long times.

According to the investigations of the last twenty years, it is certain that there is no actual destruction of the peripheral parts in Waller's "degeneration" (see also Berblinger); for even though there is complete destruction of the axis cylinder and the medullary sheath, there is nevertheless an increase in the number of the nuclei in Schwann's sheath, and a change of the sheath into a plasma-rich nucleated structure; in later stages, this shows striped cell bands in the protoplasm which are believed to be formative cells for the nerve fibrilla (see autogenetic theory p. 2020), which we will again encounter in speaking of regeneration (p. 2020).

As in many points in nervous pathology, these changes in the peripheral "degenerated" parts of the nerve are subject to many explanations; thus, according to Durante (lit.), there is only a progressive disappearance of the differentiated substances, *i.e.*, the fibrillae of the nerve fibers, which then appear as protoplasmic elements in the form of bands, and heavily stained cells (regression cellulaire), which remain in this condition for a long time or undergo various changes. E. Neumann believes that Waller's "degeneration" is a de-differentiation of the nerve fibers (axis cylinders and myelin), to an embryonal neuroplasm, as a result of the retained communication with the central ganglion cell, which has a regulating influence on the entire chain of nerve tissues. Durck also believes completed Waller's degeneration is a change of the nerve fibers into nucleated neuroplasm cylinders (for more details, see under regeneration, p. 2019 and forward).

Gross changes in the central stump are shown only as a degeneration reaching backward beyond several of Ranvier's nodes; the finer changes which develop in the central parts up to the nucleus itself have been discussed under retrograde degeneration p. 1873; their significance, however, is still obscure.

After section of a nerve, degeneration proceeds centrifugally along its entire course and in all fibers, but the sensory fibers degenerate more quickly than the motor (Monckeberg and Bethe). After crush wounds, on the other hand, the fibers degenerate by degrees.

Soon after section there may be demonstrated histological swelling of the myelin in the medullary sheaths, and after two to four days, destruction into clumps, drops and globules. There then occurs a chemical decomposition of the myelin; see Spiegel, concerning the physical changes in the myelin sheath in the beginning of Waller's degeneration, such as a loss of the doubly refractive power and a swelling which renders easier the removal of the debris; the stearic acid which appears, stains black by Marchi's method (p. 1874). Fat granule cells remove the products of destruction in weeks to months. With the destruction of the nervous tissue, there are also swelling, vacuolation and crumbling of the axis cylinders; indeed, according to Monckeberg and Bethe, the latter are the first to undergo changes (Kimura). The nuclei of Schwann's sheaths grow vigorously; the sheaths become protoplasmic nucleated bands. These growing Schwann cells become phagocytes, and as fat granule cells, help remove the destroyed material (Doinikow); as long as this has not been completely removed, particles are retained in Schwann's sheaths. After complete resorption, the parts remaining are the overgrown and changed Schwann's sheaths and the connective tissue sheaths of the nerves; if the latter undergo growth, there is fibrous induration. If this growth does not take place, the nerve appears gray.

Simple degeneration of the peripheral nerves (and muscles) occurs, furthermore, after destruction of the motor ganglion cells of the anterior horns of the spinal cord and of the motor roots. Corresponding to the successive destruction of the motor nerve cells, cross section of a nerve shows entirely healthy or mildly changed fibers alongside of those which have been totally degenerated. After destruction of the end apparatus in the muscle itself, there may be (functional) atrophy of the corresponding nerves just as, for example, degeneration of the optic nerve follows enucleation of the eye. Senile atrophy also occurs in nerves. When it progresses very slowly, the nervous substance disappears gradually, whereas the axis cylinder in the destroyed medullary sheath may be retained permanently, or for a long time. The nerve becomes small, gray. glassy (gray degeneration). Corpora amylacea may also appear (p. 1878),

Neuritis or True Inflammation and Hematogenous Degenerations. The designation neuritis is just as indefinite in determining or describing actual inflammations of the nerve as in describing degenerations of peripheral nerves which have been brought about by hematogenous toxic substances. In acute actual neuritis the nerve is red and swollen, and

ofttimes spindle-shaped. The vascular connective tissue part, *i.e.*, the epi- peri- and endoneurium, is the site of an exudative inflammation; hyperemia, entrance of fluid exudate and leucocytes are characteristic. Hemorrhages and pus formation may also occur. Mild inflammations may be confined exclusively to the interstitial tissue and may heal completely. More serious degrees destroy the nerve and perhaps the axis cylinders.

In suppuration and gangrenous inflammation the nerve disappears entirely; cicatrization then follows. The suppurative inflammations of nerves by continuity from the neighborhood, *e.g.*, a phlegmon or an infected wound, or suppurative arthritis, or sphenoid or temporal caries to the facial nerve, etc., occasionally extend centripetally (ascending and migrating neuritis, lit. Bolten).

After the nerve fibers disappear in acute and chronic neuritis there is a more or less vigorous connective tissue formation. If it is marked (proliferative neuritis), the nerve becomes thicker, hard, and usually gray; it may, however, be reduced to a pigmented or gray white, flat strand, blending with the surrounding overgrown connective tissue. Occasionally there are focal nodular fibrous swellings (disseminated nodular neuritis). According to this spread, the clinician differentiates a localized and a multiple neuritis (polyneuritis).

Among the causes of hematogenous nerve degeneration and inflammation may be mentioned: intoxications and infections, traumata, circulatory and nutritional disturbances. Often, however, a definite cause cannot be found and the term "exposure" is used, which, at least, may be of predisposing importance.

Among the infectious diseases during the course of which a multiple, or a localized neuritis occurs, may be mentioned: typhoid fever, typhus fever, pharyngeal diphtheria, scarlet fever, whooping cough (Clauss), variola, erysipelas, influenza, acute articular rheumatism, pneumonia, malaria, tuberculosis, gonorrhea (Lesser), syphilis (the latter doubtful, according to Nonne). In the pyemia of the puerperium there may, for example, be a neuritis in the nerves of the arm (Kast). A diphtheritic paralysis is the most frequent form of polyneuritis occurring on an infectious basis (p. 613). This neuritis is usually due to the effect of toxins, but in a certain number of cases is possibly due to localization of the infection. According to Stahl (lit.), the peripheral character of the neuritis as a primary condition is quite improbable, and he showed that the symptom may occur from a primary inflammatory process in the spinal roots.

Beri-beri, a disease occurring particularly in Java and Japan, but also in other countries such as southwest Africa (Dansauer), is an endemic form of multiple neuritis (rice polyneuritis), which was thought to be due to an

infection (Baelz, Scheube, Daniels), or an intoxication from certain fish (Miura, Grimm), or poorly preserved (Yamagiwa) or polished rice (Segawa, lit.); lit. by Durck. According to Glogner, beri-beri is a "polymyositis" with segmentation. See also Nocht, Schaumann. The tendency is now to regard beri-beri as a purely alimentary and not an infectious or toxic disease, called forth by the eating of polished rice; it is considered an avitaminosis (p. 2003); the covering of the rice contains the vitamin, a nitrogen-containing body like nucleic acid (Funk). This substance also cures the disease. Heating to 120° C. destroys the vitamins (Tsuzuki, Schnyder, lit., Schuffner, Schilling, lit. by Kimura, Nagayo and Schaumann). Seemingly spontaneous cases of multiple neuritis in individuals with no infectious disease are of unknown, but possibly infectious nature.

Among the intoxications might be mentioned: chronic poisonings with alcohol and with lead. Acute intoxications with arsenic, carbon monoxide, mercury and others, as well as with ergot, are considered. In diabetes mellitus, carcinoma, chronic constipation, putrid bronchitis, polyneuritis may occasionally appear perhaps due to autointoxication. Alcoholic neuritis is the most frequent form of polyneuritis; it practically always runs an acute or subacute course; exposure to cold, or a disease with fever, often determines its outbreak. The legs are usually involved first; the peroneal nerves, as a rule, are symmetrically paralyzed (foot drop). There are no bladder difficulties, but the central nervous system is occasionally involved also. Concerning the question if in alcoholics there is destruction of the medullary sheaths without inflammatory changes, see Fahr, who believes this is possible. Lead paralysis occurs in chronic intoxications with lead in the form of: (*a*) metallic lead, in type setters (lead-containing dust) and type pourers, (*b*) white lead (basic lead carbonate) in painters, (*c*) oxide of lead in china and porcelain and cooking utensil workers. The paralysis is confined almost always to a particular peripheral muscle territory, *i.e.*, to the extensors of the hand and the finger; it is usually bilateral. Only muscles supplied by the radial nerve are attacked; the supinators and the triceps are not involved in typical cases. The median nerve and the ulnar nerve which supply the small muscles of the hand may also be paralyzed. The hands are bent and swollen; when they are passively lifted, they fall back into the bent position. Those nerves are particularly involved, the muscles of which undertake the hardest activity (Teleky, lit.). Exceptionally, but particularly in children, the paralysis involves the lower extremities through the peroneal nerve with sparing of the anterior tibial muscle. The muscles atrophy. Lead colic often precedes the appearance of the paralysis; degenerations have been found experimentally in the ganglion cells of the intestinal wall (R. Maier). A number of the cases of lead paralysis are of spinal origin,

following anterior poliomyelitis (Oppenheim); see also p. 551. Arsenic paralysis usually occurs in acute intoxications. The so-called puerperal neuritis (Moebius), which appears in the latter half of pregnancy (von Hosslin) or after the puerperium, and in its milder form is usually localized particularly in the median and ulnar nerves, is probably also an auto-intoxication (Eulenburg), but there is a "leg type" as well as a severe general puerperal neuritis.

The origin of traumatic neuritis is very varied. Wounds, pulls, crushes, by force of muscle action, pressure or the ingrowth of tumors, etc., lesions from dislocated parts of joints, fragments of bone, pressure of a callous, among other things, must be considered. Among the nerves of the extremities, the radial nerve is most frequently attacked.

Among the circulatory and nutritional disturbances which produce multiple neuritis might be mentioned severe forms of anemia, and senility; in the latter probably arteriosclerosis is the important factor.

See Wertheim-Salomonsen, concerning the histopathology of neuritis, and particularly the question of the regenerative processes; Doinikow found buds (thin non-medullated fibers) which arose as a regenerative effort on the part of intact fibers.

(*Appendix:* amyloid degeneration in nerves involves the epi- and endoneurium, and the blood vessels of the peripheral, but also of the sympathetic, system including sympathetic ganglia; according to Belokrenitzky (lit.), it is not rare, but has been little studied.)

Infectious Granulation Tumors

Tuberculosis and syphilis of the nerves are seen chiefly in the cerebral and spinal nerves; the changes follow tuberculous or syphilitic processes in these regions, from which they spread and lead to atrophy (see under brain and spinal cord, pp. 1916 and 2011).

Leprosy is frequently localized in the nerves and produces a well known nervous form with anesthesia (see leprosy, under skin). The lepra bacilli which invade the nerve connective tissue, provoke a growth of granulation tissue, which leads to degeneration of the nervous parts, and later, to spindle cell fibrous thickenings of the nerves. See Askanazy (lit.), concerning the relationship of the skin nerves in leprosy of the skin, and the exceedingly important role of the nerves in leprosy as a whole.

Regeneration

(See Berblinger and Kimura for lit. concerning this complicated question.)

When a peripheral nerve is sectioned, the entire distal part degenerates to the endings in the muscle; destruction attacks first the axis cylinders,

but soon appears in the myelin sheaths. The degenerated portion loses its properties of conduction and electric irritability. In the central stump, the degeneration spreads proximally to the next Ranvier's node, finally, a few segments higher. If the ends of the nerves are reunited, a granulation tissue grows from the connective tissue parts of the nerve and later becomes a cicatrix. Nervous communications may be reestablished, whereby the regenerative fibers penetrate through the scar. The opinions concerning these processes are still divided. There are two main theories: (1) The centrogenous or the outgrowth theory (Waller) assumes that the degeneration in the peripheral part is a consequence of the disturbed communication with the ganglion cell which is the nutritive center of the nerve; during regeneration young nerve fibers grow independently from the central (proximal), retained stump of the fiber, *i.e.*, without participation of the peripheral elements. The regeneration is brought about, therefore, by sprouting of the central stump.

According to this outgrowth theory, the young nerve fibers penetrate the severed nerve ends which are jointed by connective tissue (Waller); they pass through and grow like seedlings into the degenerated peripheral (distal) part; since they are in the epineurium and perineurium, but also, in part, in the old neurilemma tubes, they gradually reach the end apparatuses (Vanlair, Stroebe, Perroncito, Berblinger, Kimura and others), and thus the connection is re-established between the ganglion cells, the nerve fibers, and the end apparatus. The end plates of the motor nerves lying in the muscles are regenerated in the muscles themselves (Gessler). Thus, there is no reunion of the nerve fiber by growing together of the peripheral and central parts, but a new formation of nervous tissue into the peripheral part from the central stump. Newer investigations assign an important role at the situation of the section to richly nucleated plasma bands, arising from Schwann's cells; these bands appear not only in the central but also in the peripheral end, and attempt to unite the separated fibers through the scar; these cell strands, according to the centrogenous theory, constitute the plasmatic growth path for the newly formed nerve fibers arising from the central ends (Berblinger).

According to Vanlair, the degenerated parts are only a path or a bridge; if the separated ends are not closely approximated, or if a piece is excised, the interposition of an indifferent material (pieces of rubber tubing) may serve the same purpose (Gluck). The excised piece dare not, however, be longer than 4 centimeters (Wolfler). Edinger's bridge method with agar-filled formalin hardened arteries from calves has proved impractical (Wollenberg, Enderlen and Lobenhofer); see also the method of bridging devised by Berthe. If a piece of nerve from one defect is implanted into another, it is used only as a path for the younger fibers

growing from the central end, and of itself takes no part in the new formation of fibers (Huber). According to Perroncito, collateral paths may occasionally function without anatomic regeneration.

The formation of new fibers in the proximal end begins by a swelling of the axis cylinders over a certain length of the nerve above the situation of section; they then separate; buds appear, which still retain irregularly thick medullary coverings inside of Schwann's sheaths and therefore appear varicose. The young fibers produce entire bundles, which make their exit from Schwann's sheaths and acquire a neurilemma sheath. The return of function after suture of a nerve may take a long time, according to Spielmeyer, even up to two years.

2. The autogenetic theory (cell chain theory). According to this, central processes play a role in regeneration, but the new fibers which re-establish the continuity, arise from Schwann's cells of both the central and the peripheral stump. This occurs after the degenerative changes have practically ceased. Those fibers arising in the peripheral stump then join with those from the central stump.

Because of the various opinions concerning the details of this process, we must call attention to the careful experiments of Durante, and state merely that, according to his opinion, regeneration takes place from Schwann's cells (v. Bungner, Neumann, Wieting, Spielmeyer and others). These are the cells mentioned on p. 2013, which were formerly believed to be connective tissue cells, *i.e.*, of mesoblastic origin (Stroebe), but which are now believed to come from ganglion anlage, *i.e.*, of ectoblastogenic origin; they are called neuroblasts, neurocytes, or nerve fiber cells with the ability by growth and differentiation of their protoplasm to produce "discontinuous" axis cylinders and even medullary sheaths. The neuroblasts become elongated, disappear to richly nucleated bands (cell chains or cell bands), and where they come into communication with the parts of the proximal stump, favor functional passage of conduction and differentiate into bands of neurofibrils; in the other case, *i.e.*, when union is not completed, this further differentiation does not occur, because, according to Neumann, it is the communication with the central nerve stump which gives the stimulus to this process (see also Bethe). According to A. Kohn, ganglion cells, as well as nerve fiber cells—cells of Schwann's sheaths,—come from a general source, the neurocytes of the embryonal nerve; the same structures can also produce glia tissue. O. Schultze showed that the peripheral nerves could come from primary cells, the neuroblasts. S. R. Cajal, one of the founders, and consequently a believer in the neurone teaching, Perroncito and others, could not, however , convince themselves of autogenous regeneration, but believe rather that the neurites (axones) of the central stump are independent of

all cell change, and can produce new fibers, which branch and grow with great rapidity, even the second day after the injury, long before anything is seen of the cell bands described by v. Bungner; these new axones force their way peripherally in masses and some reach the peripheral stump, in which they sink; and separate its fibers. Regeneration begins after a few days and takes a variable length of time. Very peculiar and not easily correlated with this Vanlair-Stroebe idea is the fact, that after nerve suture, the function reappears the more slowly and the more incompletely, the nearer to the center the injury to the nerve has taken place (Oppenheim). With the nerves over the joints of the hand, it often takes only three to six weeks.

After section of a peripheral nerve end as in amputations, the connective tissue of the central stump grows, and nerve buds advance into this granulation tissue which rapidly becomes a cicatrix. In every old Schwann's sheath, there are many newly formed nerve fibers. Since they cannot run a straight course in the scar tissue, they become much convoluted and ofttimes appear in such large masses that they produce a tumor-like, wedge-shaped swelling, a so-called amputation neuroma, as a rule, embedded in the scar of the amputation stump and tightly adherent to the skin.

In the beginning without medullary sheaths, the nerve fibers during the course of time (months) come to retain a tender myelin covering. Neighboring amputation neuromas may coalesce with one another. But neuromas may also appear centrally and produce a string of pearl-like appearance. The peripheral end of the stump shows a basket-like thickening from increase of nervous connective tissue and growth of Schwann's cells, but this cannot be designated a peripheral neuroma (Berblinger).

These neuromas, as well as the pressure of scars, are said to be related to the development of trophic ulcers, inasmuch as they irritate the sectioned central end of the nerve, an irritation which is spread centripetally, and leads to disturbance in the tonus of the sympathetic and thus to vaso-motor trophic disturbances. At least, disappearance of the trophic disturbances has been seen to follow removal of the neuroma, *i.e.*, after neurolysis (Bruning, lit.). [For morphologic changes in nerve cells following axon injuries, see Nicholson, F. M., Arch. Neurol. & Psych. 11: 680: 1924. For remote results of operations on peripheral nerves, see Platt, H. and Bristow, W. R., Brit. J. Surg. 11: 535: 1924.—*Translator.*]

Tumors

Primary Tumors of the Nerves. False Neuromas.—An isolated tumor on a nerve with the character of a fibroma or a fibromyxoma is by no means rare. These have been called for many years "false neuromas"

or fibromas, fibromyxomas, neurofibromas, neuromyxomas, etc. Thus, isolated as a rule, spindle shaped, painful fibromas appear along the trunks of the larger nerves to the extremities, thus, *e.g.*, in the axilla (Schmidt and Delblanco), and particularly often in the median nerve (fig. 980). These tumors are certainly closely related to neurofibromatosis. Circoid neuromas (p. 2026) are also false neuromas. Under the term "skin fibromas," appear many which have been called neurinomas (p. 2029), or tumors composed of both nervous and connective tissue parts, so that differentiation into true and false neuroma is not always possible. In exceptional cases they become as large as a child's head. Lipomas also occur, occasionally multiple, and occasionally diffuse (Durante). A rhabdomyoma has been described by Orlandi (very rare). Sarcomas, usually of the spindle cell or myxo-variety, may originate from the connective tissue of the nerve or develop as a richly cellular malignant variety of a neurofibroma (p. 2028 and also p. 2030).

FIG. 980.—Spindle-shaped fibroma of median nerve (isolated neuroma of trunk); nat. size.

Isolated tumors of the optic nerve before its entrance into the bulb, lead to spindle shaped swellings, diminution in vision, pain and exophthalmus, usually in younger individuals; some call them fibromatoses, with a tendency to malignant change (see Byers); others call them gliomas (Harbitz), originating from the glia in which the optic fibers are embedded, and these authors separate them from the ordinary fibromas of the nerves.

The so-called cerebello-pontine angle tumors, called by others acoustic tumors, were described on p. 1936; a minority belong to the false neuromas but most of them, are of the nature of neurinomas (p. 2029). They may constitute one of the symptoms of neurofibromatosis (Henschen; see also Rossle).

True Neuromas. There may be differentiated: (1) simple, purely fibrillar neuromas without ganglion cells, and (2) cellular ganglion neuromas. All true neuromas date back to embryonal irregularities and cell inclusions, and particularly to multipotent embryonal neurocytes; in their diverse development they produce: ganglion cells, glia cells and nerve fiber cells (Schwann's and capsule cells and ganglion cells). In the sympathetic nervous system it is believed that the development comes from indifferent cells, which arise from the embryonal spinal nerves (embryonal neurocytes of the spinal nerves) (A. Kohn); see Pick and Pick-Bielschowsky.

Occasionally we meet solitary, extremely painful, cutaneous, or more frequently subcutaneous, neuromas (tubercula dolorosa), particularly in

the extremities. By Weigert's hematoxylin stain it is often possible to demonstrate many medullated nerve fibers in the neuromas arising from peripheral nerve branches; non-medullated neuromas, on the other hand, are histologically difficult to recognize (differential diagnosis on p. 1650); there also occur neurinomatous forms of "tubercula dolorosa" (see fig. 986, p. 2029); the author examined such a tumor adherent to a cutaneous nerve; it was as large as a pea, and came from the thigh of a 56 year old man.

True neuromas in the form of ganglion neuromas (mature, Pick) are very rare; they are usually composed of rounded ganglion cells and pre-

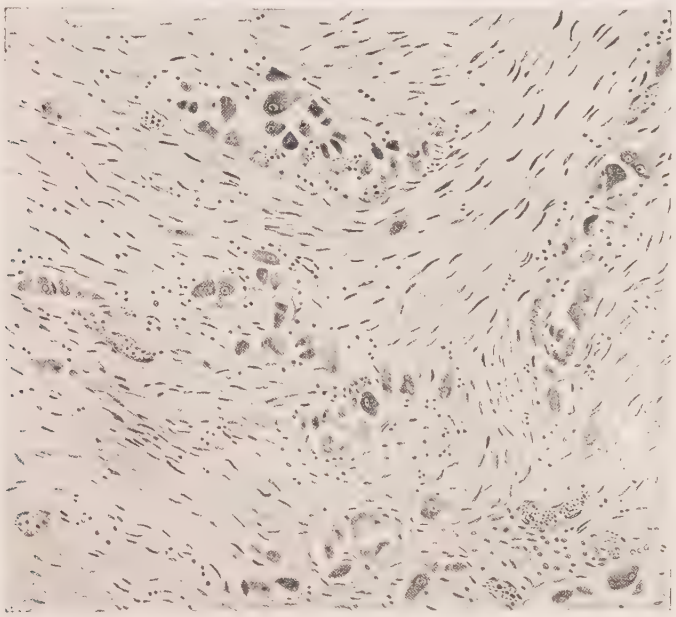


FIG. 981.—Ganglion neuroma of adrenal (sympathetic), girl 9 years old; tumor as large as a hen's egg. Masses of ganglion cells are seen, partly in nucleus-containing envelopes; some cells contain several nuclei, some have prolongations. Ground tissue composed of non-medulated nerve fibres and blood vessels. (Medium power.)

dominantly non-medullated nerve fibers, along the cerebro-spinal nerves (Czerny, Benda, Verocay and others); but the great majority comes from the sympathetics, usually from the left border strand (Wegelin, anatomy by L. R. Muller), or from the medulla of the adrenals (see sympathetic tumors p. 1287); some come from the larger abdominal ganglia (coeliac ganglion, Beneke); but they also come from the ganglion of the cervix of the uterus (Beneke), more rarely, multiple forms appear in the subcutaneous tissue (Knauss, Kredel-Beneke) and even in the capsule of the knee joint (Hagenbach). These tumors have reached the size of a child's head (comprehensive lit. by Wahl). A case described by Beneke and one by

Miller represent malignant ganglion neuromas, with metastases containing ganglion cells and nerve fibers in the lymph nodes. Sommerfeld believes these "metastases" are primary tumors, since it seems to him improbable that a ganglion cell tumor should metastasize; nevertheless Berner believes that it is possible for true metastasis to take place; see literature in appendix.

Marchand described a purely cellular tumor of the Gasserian ganglion with infiltration of the trigeminal nerve and its branches, which he believes came from an undifferentiated stage of a ganglion anlage, and which he calls a neurocytoma (neurocyte, p. 2020); according to Pick, the purely cellular neurocytomas of the spinal ganglia belong to these "immature" neuromas. In the case of Risel, the glia had also developed. For other tumors of the Gasserian ganglion (glioma, sarcoma, endothelioma), see Giani, literature.

FIG. 982.—Neurofibromatosis of intercostal nerves from a case of general neurofibromatosis with participation of peripheral, cerebral, spinal and sympathetic nerves. Tumor formation (glioma) and softening in spinal cord; boy 15 years old; ½ nat. size.

Under the name of **neurofibromatosis** (v. Recklinghausen's disease), there are considered multiple fibromas of the nerves, mollusk-like skin tumors, pigmentation of the skin, and elephantic thickenings, isolated or in groups. To the disease picture are usually added other malformations, anomalies, and tumor formations, thus gliomas of the central nervous system (endotheliomas of the dura). The various components of the picture of neurofibromatosis are presented as:

(*a*) Multiple fibromas of the nerves, (neurofibromatosis in the narrower sense), chiefly in the spinal nerves, either the deep lying branches (ischiadicus and its branches, vagus) or skin branches, but the cranial nerves and the sympathetics are not immune. There appear multiple, nodular, spindle shaped, rosary-like, or diffuse, earth worm or circoid, plexiform swellings in many nerves, some of small degree, others as large as a fist, at times on almost every one (fig. 982). The growth is established in the region of the endo- and perineurium, splits the nerve apart or surrounds it, or involves it tangentially, almost passes the basket-shaped swelling (as in fig. 980) or crosses it. Nerve fibers may be retained within

(fig. 983). The plexiform tumors are usually of tougher consistency. Functional disturbances, especially motor, are relatively rare, but sensory disturbances are common.

Other anomalies and malformations, such as epispadius, crypt-orchidism, polythelia and others are quite frequent (Adrian). Tumors in the spinal cord such as gliomas, and tumors of the brain and spinal meninges may be combined (lit. by Kaulbach and Verocay). Combinations with hypernephromas were seen by Kawashima, Saalmann. Concerning changes in the bones, see Stahnke, (lit.). Tumors, particularly

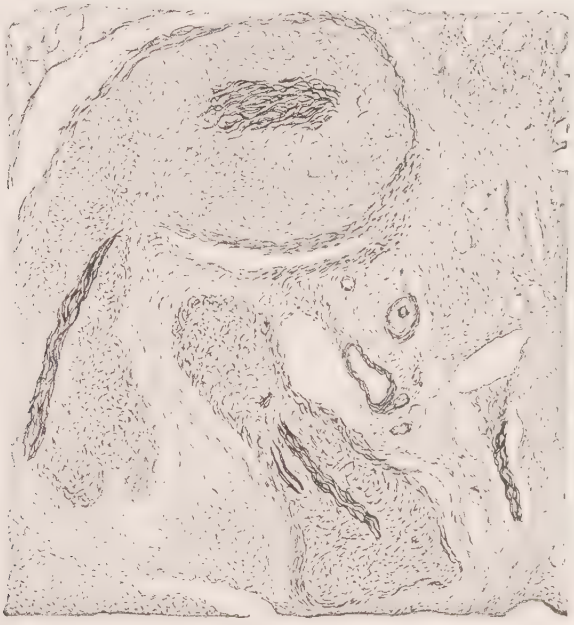


Fig. 983.—From an elephantic plexiform neuroma of left parietal-occipital and cervical areas of girl 19 years old. The tumor was observed for 10 years, growing rapidly toward the end. Nerves are seen; the loose mucous-like tissue proliferation begins from the outward endoneural as well as from perineural parts. Nerve fibres (dark, wavy) are partly preserved as united strands, partly fringed out. Between nerves the loose cellular perineurium contains fatty tissue and vessels. (Very low power.)

in the smaller cutaneous nerves, are very frequent; indeed in Harbitz's observations they were always present, so that multiple skin tumors are the rule, (fibromata mollusca, fig. 985). Pigmentation of the skin is also frequent; according to Thomson (lit.), in 25% of the cases; according to Soldau, these discolorations correspond to a fibromatosis of the end fibers of the cutaneous nerves. Very often there are congenital spots and naevi, which increase in intensity and number as time goes on. These are buds or larger oval spots, or merely diffuse brown staining. The buttocks, the neck and the face are the favorite sites.

(*b*) A plexiform neuroma (v. Bruns), is a so-called nerve fibroma, in which a locally developed convolution becomes worm-like or tuft-like, occurring particularly in the skin and the subcutaneous tissue in a circumscribed region of a nerve distribution. This form of tumor is practically always congenital. Sites of preference: the upper lid, the cheeks, region of the ear, the back, the breast. Large, plump, or pendulous, lobulated skin thickenings are called elephantiasis neuromatosa or pachydermatocele (fig. 984). Even in these cases, relationships to nerve connective tissue may be demonstrated. Occasionally, deformities are produced, particularly in the feet, legs, external genitalia, the nape of the neck, the face, etc., finally the entire integument. (Details by Cassirer; see also Hildebrand.) The elephantiasis is usually congenital, or appears in early childhood, as a slow, painless growth. The skin covering the parts is tough, raw, rough and folded. Later the elephantiasis may become stationary. These may be called circoid neuromas or plexiform neuromas or racemose nerve fibromas (lit. by Strauss); others call them neuromas (Verneuil) in the true sense, and the idea that there is not only elongation but also increase in the nerves in addition to the connective tissue growth, is very often met. But Wegelin could not demonstrate a new nerve formation; even the rows of elongated and rod-shaped nuclei which are believed by Verocay to be Schwann's nuclei (see under neurinoma, p. 2029), are considered by Wegelin as nuclei from the connective tissue of the nerve sheath. Wegelin holds that these circoid neuromas are in reality hamartomas (see opposed, Hoffmann).

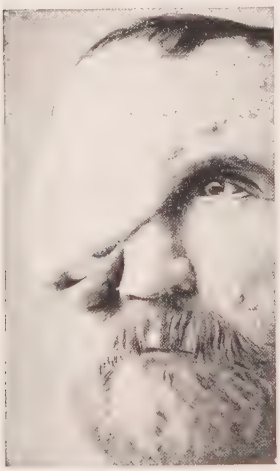


FIG. 984.—Lobed elephantiasis of r. upper and lower lid; various small nodules on forehead and cheeks of man 45 years old.

(*c*) The cutaneous fibromas of the nerves, also called neurofibromas or multiple skin fibromas, have an entirely different appearance; according to v. Recklinghausen, they are fibromas in the smaller branches of the sensory cutaneous nerves. There occur pure fibromas of the peri- and endoneural connective tissue; others show a small number, still others show a larger number of nerve fiber cells (see under neurinoma p. 2029). These tumors of microscopic size to the size of a man's head, often occur in enormous numbers, and are situated deep in the skin or subcutaneous tissues, or are like warts or nodules, or semi-circular, flat, pedunculated, or pocket-book, or polypoid, structures on the surface of the skin, mostly distributed on the trunk (fig. 985), the neck and the head, in regular or asymmetrical distribution. As a rule, the consistency is soft, (fibroma

molluscum), although exceptionally it is very hard; on pressure they are somewhat painful. They are sometimes present at birth. Harbitz discusses the possibility of partial disappearance of the skin nodules. The name elephantiasis molluscum is used to describe large irregular nodules arising from isolated nerves.

Etiology and Development of Neurofibromatosis.—It is now fairly well agreed that these various forms are related, and are symptoms of a congenital disease of the anlage, *i.e.*, they are malformations in the broad

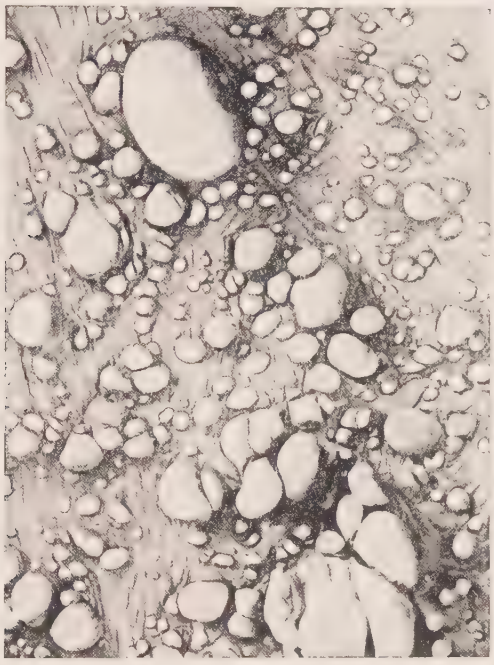


FIG. 985.—From skin of back of a case of neurofibromatosis (v. Recklinghausen's disease). Tumors attached both by broad bases, and by pedicles; ½ nat. size.

sense of the word (Thomson, lit., Adrian, lit., Harbitz, lit., and others). Harbitz believes the disease is a general constitutional condition, in which anomalies occur in the osseous system, disturbances in intelligence (brain changes, see Nieuwenhuijse), infantilism, etc.; it is a developmental anomaly in the broad sense of the term with a tendency to "connective tissue" tumor formation (fibromas, myxofibromas, sarcomas) in particular organ systems (nerves, skin). The changes are present at birth, or appear from latent anlage, even in later decades. The beginning is usually evidenced by pigmentation and the formation of tumors in the skin. Pigment anomalies of the skin are quite frequent; anemic naevi (Naegeli), adenomata sebacea of the face (Nobl) and other kinds of naevus-like structures are common. Glioma and tuberous sclerosis of the brain have

also been observed (Orzewski and Nowicki). In addition to the cerebral nerves, the spinal branches (the spinal roots, in the case of Wallner) may be thickened and nodular. The sympathetic nerves may also be involved (Verocay, Harbitz, Pick, Herxheimer-Roth). Gastrointestinal nodules may be found; some become sarcomas and produce symptoms, *e.g.*, stenosis of the pylorus; otherwise, as a rule, they are symptomless. Verse saw growths in the connective tissue of the pelvis producing obstruction in the vagina. Adrian found an heredity history in $\frac{1}{5}$ of the cases. Harbitz described congenital elephantiasis in five generations. The best evidence for the relationship of the various forms is the fact that the disease shows all kinds of variations in different generations. The male sex seems to be attacked more frequently. (Lit. concerning neurofibromatosis in the appendix.) Of particular interest is the more or less frequent transformation into malignant tumors of spindle cell, myxo cell or giant cell types, described as richly cellular varieties of fibromas of the nerves (v. Winiwarter, Westphalen, v. Bruns, Garre, and others). The forms of neurofibromatosis behave differently in this respect, for practically none but those described under (*a*) as multiple fibromas of the nerves undergo sarcomatous transformation. According to v. Bruns, $\frac{1}{12}$ of all these cases die of sarcoma (lit. by Thomson, Adrian, Hoekstra). In Basel, the author saw a girl with multiple neurofibromas and but few tumors of the skin; she developed a colossal spindle cell sarcoma in the ischiadicus. Metastases are rare, but one nodule after the other becomes sarcomatous.

Newer Ideas Concerning the Histogenesis of Neurofibromatosis.—Durante, followed by Francini (lit.), proposed a new and uniform viewpoint concerning the nature and origin of most nerve tumors. He begins with the idea of regarding the entire interannular segment, *i.e.*, between two Ranvier's nodes, as being composed of highly differentiated cells, corresponding to the theory of the multicellular composition of nerve fibers, or the cell chain theory. He believes that there is regression of the segment cells of the nervous tubes, until the differentiated parts of the segments disappear (axis cylinder and myelin), while the indifferent protoplasm increases and allows nuclear division and the appearance of homogeneous, nucleus-containing bands, or spindle shaped cells (neuroblasts). The spindle cells may coalesce, produce new bands of protoplasm, and become medullated or non-medullated nerve fibers. Thus there appear amyelin and myelin neurones. If these young cells grow as separate spindle shaped elements, there arises a cellular neuroma or a cellular neuroblastoma, which is called a sarcoma of the nerves. On the other hand, these newly formed nervous tissues are able to undergo certain metamorphoses; when they undergo atrophy, they take on the appearance

of connective tissue, or inbibe mucin or take up fat droplets and become similar to fibromas, myxomas, or lipomas of the nerves, but their fundamental nature is always that of a neuroma. Antoni disagrees with this theory.

The neurinoma teaching of Verocay disputes the purely fibromatous nature of neurofibromatosis; Wallner (lit.), in an interesting historical essay, points out that this theory has many adherents. Verocay describes a diffuse and circumscribed increase of the cells of Schwann's sheath (nerve fiber cells, p. 2013), which he believes are the forerunners of the tumor, but he does not entirely deny the possibility of a connective tissue

FIG. 986.—Neurinoma, from a spindle-shaped tumor of neck the size of a pigeon's egg; one of similar size in pectoral muscle; removed by operation; servant girl 29 years old; clinically called v. Recklinghausen's disease. The wavy nucleated fibrilla bundles are seen with the nuclei in many instances arranged parallel. (Medium power.)

part in neurofibromatosis. In a second work, he expresses the opinion that the tissue of multiple nerve tumors is not connective tissue since it does not stain red by van Gieson's method, but yellow; it is rather a peculiarly nucleated, finely fibrillar, neurogenous tissue, composed of plexiform bands or bundles with palisade-like, parallel arrangement of the nuclei ("parade," Askanazy), and fine fibers arranged in bundles like glia fibers, or other neurogenous fibers but different from connective tissue. The antecedents are nerve fiber cells (Schwann's cells) themselves, or corresponding embryonal cells originating from neurocytes; Verocay proposes the name neurinoma for these "fiber tumors of nervous nature" without axis cylinders and medullary sheaths. Askanazy also considers

the nucleated bundles of fibers as incompletely differentiated descendents of neurocytes, but believes that it has not been proved that these fiber bundles are of nervous or of glious nature. Therefore the term "immature neurocytoma" (Pick-Bielschowsky) or "immature neuroma" is also used. Antoni characterizes the neuroma as the tumor form of the specific interstitial substance of the peripheral nerves and he differentiates a fibrillar, richly nucleated type with elongated bundles, and a reticular type which is arranged in an apolar way and has less nuclei. Antoni believes the neurinomas come from the cell material of ganglion ridges, a material which can produce ganglion cells and Schwann's cells, but not glia. By increases in the number and the polymorphism of the nuclei, there appears, according to Verocay, the neurinoma sarcomatodes. Tumors in which the connective tissue plays a greater part and dominates the structure, may be called, "mixed tumors," "fibroneurinomas," or "neurinofibromas."

Verocay has unquestionably clarified our knowledge of neurinomas, but to apply these generalizations to v. Recklinghausen's disease, (see also Herxheimer, Adami and Nicholls) and to call it merely a neurinomatosis (Wallner), is going too far, since there undoubtedly does occur a pure type of fibroma in neurofibromatosis or neurofibroma, as emphasized particularly by Harbitz, and confirmed by Wegelin (case of circoid neurinoma), the author, Ribbert, Verse, and Antoni. It is true that Pick-Bielschowsky, and particularly Herxheimer-Roth, (lit.), have verified the generalizations of Verocay, and they believe that the primary factor in nerve tumors is a growth of Schwann's cells; there is, however, a secondary connective tissue growth, and the primary growth of cells of the sheaths may be more or less completely inhibited by the connective tissue growth to practically entire disappearance. Thus v. Recklinghausen's disease at times shows a peculiar specific formation of neurinomatous tissue, and at other times, typical connective tissue. In the latter case, the term neurofibroma must be used. When there is a mixture of both tissues, which Verocay also noticed, the term neurofibroma, or neurinofibroma may be used. See Askanazy, Berblinger as well as Antoni. For the complete clarification of the question of neurofibromatosis, it is necessary to undertake further investigations and comparisons of many cases and of every possible situation in any given case. Neurinomas occur occasionally in the sympathetic system (Pick), as well as in the central nervous system. See Askanazy, concerning diagnostic difficulties in cases which are masked under the picture of multiple endotheliomas of the dura, or, as solitary tumors of the posterior pharyngeal wall. For neurinomas of the spinal cord, see p. 2011. See Schmincke, concerning the formation of neurinomas in the appendix.

Secondary Tumors.—Carcinomas occasionally invade the nerves by continuity, *e.g.*, in the parametrium in uterine or rectal carcinoma, and spread between the nerve bundles, which gradually undergo atrophy. In general, the nerves are quite resistant to the invasion of tumor tissue; they are rather compressed than infiltrated by tumor masses (in the perineurium). The tumor cells spread readily by way of the lymph channels (Schroeder van der Kolk, Colomiatti, Stilling, Ernst, see also

Fig. 987.—Cross section of brachial nerve infiltrated with squamous cell carcinoma from forearm. The carcinoma cells spread in the peri- and endoneural lymph clefts. Gland-like pictures are due to lining of lymph spaces. (× about 150.)

Moschkowitz, Basten), and the epithelial cells in the spaces produce gland-like pictures (fig. 987). Rossle described metastases in the peripheral nerves from a sarcoma of the pancreas. See the observation, p. 1967, concerning multiple secondary sarcomas or carcinomas of the spinal nerves inside the dural sac. The cutaneous nerves may be surrounded and infiltrated by metastatic skin carcinoma (Askanazy). See Schmitt, concerning the spread of a benign hemangioma in the nerves.

Comprehensive discussion of the pathological histology of the nerves, and the literature, by Durante in Manuel d'histologie pathol. by Cornil-Ranvier, 3rd. edition, 3rd. volume, 1907.

CHAPTER XI

MUSCLES

(Lit. of the pathological anatomy of the muscles by Durante and Lorenz; see also M. Heidenhain.)

Anatomy, Development.—Cross striated muscle fibers are composed of cross striated contractile elements, the fibrillae, and of non-differentiated sarcoplasm, in which are embedded the fibrillae collected in longitudinal bundles; these in turn are collected into the muscle columns. On cross section of a fiber, the latter appear as the so-called Cohnheim's fields. Each muscle fiber is surrounded by a homogeneous capsule, the sarcolemma. By the silver impregnation method of Bielschowsky, a tender fibrillar structure can be demonstrated in the "structureless" sarcolemma, (see Finkelnburg). Asai differentiates the sarcolemma into two layers, an outer non-nucleated connective tissue layer, and an inner layer applied to the muscle substance as a homogeneous covering; this latter is the sarcolemma in the narrower sense of the word. See Thoma concerning the frequent branchings of muscle fibers. The oval nuclei lie near the sarcolemma in the peripheral sarcoplasmic layers; they are arranged in longitudinal rows. Muscle fibers are actually cells which have grown enormously in their length, or, perhaps more correctly, a number of cells are combined in one fiber, but have arisen from a single rounded nucleated cell. The thickness of the fibers is variable; and each muscle is composed of fibers of different sizes (Schiefferdecker). According to Schiefferdecker, the cross sections of nuclei vary in individual humans. The number of nuclei is increased before birth; after birth there is a slow but steady increase in the fibers and in the mass of nuclei; there is thus growth of nuclei preceding growth of fibers. See Lipska-Miodowska, (lit.) concerning the glycogen content of the muscles which increases with increased carbohydrate ingestion.

Little attention has been paid to the pathological conditions occurring in the muscle spindles (lit. by L. Forster, Amersbach), which are peculiar spindle-shaped sensory end organs in the skeletal muscles connected with muscle sensation; contained within them is a number (according to Schaffer 1 to 13) of thin muscle fibers and nerves as well as blood vessels, all enclosed within a laminated connective tissue layer. The spindles are always found in the muscles of the trunk and the extremities, but are

lacking in the diaphragm, larynx, pharynx, esophagus and eye. They are special structures, very resistant to inflammatory processes in the muscle, and do not take part in the atrophy of inactivity or experimentally produced paralyses. On the other hand, they seem to be especially sensitive to toxins; Askanazy and Amersbach considered the degenerative changes of the spindles in Basedow's disease due to toxic causes. In paralysis agitans, Schiefferdecker found a change in the fibers confined to the spindles (see p. 542 for the changes in the parathyroid glands in this disease). Langhans described changes in the spindles in cretins.

Post Mortem Changes.—Rigor mortis is probably a coagulative process, but some believe post mortem accumulation of lactic acid causes a shift in the water content with swelling of the muscle protoplasm (v. Furth). After a short agonal period of relaxation, the skeletal muscles increase in consistency, become stiff, and fix the joints; they are less elastic, of pale light brown color, and opaque. The muscle contracted during life is elastic and transparent (see Paltauf). Rigor usually begins in the head (lower jaw, nape of the neck) and spreads from there; the release of the rigidity takes place in like manner (Nysten's law). It begins on the average two to four hours after death; inside of forty-eight hours it reaches its heighth, and gradually disappears at the end of about four days after death (Bartel). This rigor does not recur again. It also involves the heart muscle (Oberzimmer and Wacker), sometimes earlier than the skeletal muscle (for more details, see p. 71). Smooth muscle also takes part, particularly that of the middle and smaller arteries, of the stomach (pylorus), the urinary bladder, the intestines. The cutis anserina of the cadaver is caused by rigor of the arrectores pilorum muscles. (Concerning the origin of cutis anserina piloerection from the clinical point of view, see Koenigsfeld and Zierl.) Rigor involves the pupil (the smooth muscle of the iris), and produces narrowing, but only about ten to twelve hours after death. (More details by Placzek.) It does not spare muscles which were paralyzed during life. When the external temperature is warm, rigor begins earlier than in the cold, and in warm surroundings it also disappears earlier. It usually begins quite early, is well marked and lasts longer in stronger individuals who die suddenly. Rossbach saw almost immediate rigor after gunshot wounds. It is very early and marked in tetanus, strychnine poisoning, poisoning with prussic acid, cholera, and sun stroke. In fatty degenerated muscles, as in phosphorus poisoning, it is mild or entirely absent. Rigor does not occur in premature births, but in later months, it may appear intrauterine in those fetuses which are not immediately born; it even leads to difficulty in labor; or it may appear when the child dies during labor (see cases of Parkinson, Elliot, Murphy); see also Gussakow.

Congenital defects of entire muscles or parts is not rare. They are seen most frequently in the pectoral muscles (lit. by Bing and Busse).

Circulatory Disturbances

Anemia of the muscles which produces paleness and dryness, is found in the general anemia of long continued diseases, or following local disturbances; among the latter may be mentioned local compression and disturbance of the arterial blood supply. Total ischemia, rare because of the rich anastomoses of the blood vessels, is followed by necrosis. The muscles become yellow, cloudy, dry and brittle.

FIG. 988.—Hematoma of abd. rectus muscle; sagittal sect. through abdom. wall (right, outer skin, to left, peritoneum); $^{7}/_{8}$ nat. size.

Hemorrhages into the substance of the muscle as a result of tears, are observed in healthy muscles following traumata, and spontaneous rupture from tetanic contractions (in tetanus), or in diseased, easily torn muscles, *e.g.*, in typhus fever. A large extravasation into the muscle is called a hematoma (fig. 988).

See Hofstatter concerning traumatic hematoma of the masseter muscle as a typical birth injury following pull on the lower jaw or other obstetrical manipulations.

Primary hemorrhages are seen in hemophilia; secondary hemorrhages in serious infectious diseases such as typhoid fever, small pox, sepsis, spotted fever, or in intoxications, as from phosphorus.

Atrophy and Degeneration of the Muscles

Atrophy.—In simple atrophy of muscles, the normal cross striation is retained, but the fibers become smaller and may finally disappear completely. By loss of the hemoglobin contained in them, they become pale, like the flesh of fish, or dry and brown, because the fibers which are constantly growing smaller collect pigment as yellow and brown granules (pigment or brown atrophy).

Finally, the sarcolemma tubes contain no contractile substance; the interstitial connective tissue is increased in amount, as is also the number of nuclei (Askanazy) which become many shaped (illustrations by Kottmann); these are, in part, regenerative processes, but it is also believed that the muscle nuclei have resorption functions ("myoclasts").

The pigment formed is analogous to that of brown atrophy of the heart. In addition, however, there occurs a pigment which gives the iron reaction, probably derived from the muscle substance, perhaps from the muscle hemoglobin (Ishida).

Simple atrophy follows section of the corresponding nerve, after tenotomy, after adhesions, disease of the corresponding joints, and furthermore, as a result of inactivity, or of cachexia, or senility. It follows compression of tumors; according to Murk Jansen, atrophy of a monarticular muscle (passes over only one joint) may result from increased and steady tension over an inflamed joint. For more details, see Schmidtmann.

Neuropathic (Neurogenous and Spinal) and Myopathic Muscle Atrophy.—In that group of muscle atrophies which are designated as neuropathic, the foundation for the disappearance of muscle tissue is disease of the peripheral or the central nervous system. Thus neurogenous muscle atrophy occurs after section or degeneration of peripheral nerves (as, for examples, in tabes), following multiple neuritis; degenerative paralyses follow intoxications, *e.g.*, chronic lead poisoning, or lesions (section) of pure motor nerves. Muscle atrophy of spinal origin occurs after disappearance of the ganglion cells of the anterior horn and the bulbar nuclei. This was discussed under spinal cord. The extent of the affection follows closely the spread of the spinal cord disease. A particular form which follows gradual successive disappearance of the motor spinal ganglion cells with the production of an amyotrophic spinal progressive muscle atrophy (type Duchenne-Aran) has been discussed on p. 1996.

There must be differentiated from the above mentioned forms of progressive muscle atrophy, a myopathic progressive muscle atrophy, the "distrophia musculorum progressiva," or **primary myopathia.** In this condition the muscle is the primary part diseased, and changes cannot be found in the nerves or spinal cord.

The causes are not known with certainty, but in all probability this disease is due to some congenital developmental anomaly; it is now thought that it is an hereditary degenerative condition. A similar viewpoint has been reached regarding **lipodystrophia progressiva,** which Simons (lit.), believes is a sister to muscle dystrophy. There is tremendous emaciation from the disappearance of fat from the face, the trunk, the arms, while the lower part of the body (the hips, the buttocks, the legs) becomes voluminous from the deposit of fat. The muscles and nerves take no part in the process. Weitz (lit.) discusses the heredity of the condition.

Anatomic Changes in the Muscles.—Several changes are combined: the primitive fibers are partly atrophic, partly in a state of true hypertrophy (according to Oppenheim the cross section of a fiber measures 230 microns, as against 10 to 100 microns for the normal, Stohr). There are forms in which the atrophy is preceded by a hypertrophy. Spaces and vacuoles in the fibers, and amitotic nuclear proliferations can be found. The muscle fibers may also be increased in number. The internal perimysium seems more richly nucleated, more markedly developed, and fre-

quently changed into fatty tissue. This lipomatosis of the connective tissue leads to a lipomatous pseudo-hypertrophy of the atrophic muscle and is a secondary process, *i.e.*, a result of atrophy of the muscle fibers, or a primary process in which the primary development of fatty tissue compromises the muscle fibers, or, thirdly, atrophy of the fibers and lipomatosis occurring simultaneously. In isolated cases, a form of dystrophy has been observed which began with shrinking of the muscles (retracting progressive dystrophy of the muscles, see Steinert and Verse, lit., v. Werdt).

The differences between primary myopathy and spinal myopathy. The primary myopathies begin in younger individuals (the spinal myopathy as a rule, during middle life), and in many cases are an hereditary familial disease; in spinal myopathy, heredity plays no part. The disease has a chronic character. The dystrophy involves particularly the musculature of the back, the pelvic girdle, the lumbar vertebral muscles, the shoulder girdle and the upper arm and thigh. The gait becomes toddling; lordosis of the lumbar vertebrae is developed, because the muscles which stretch the vertebral column and supply the hip joint become weaker; this lordosis is corrected during the sitting posture, when the pelvis is well supported. The shoulder joints become "loose." The shoulder blades stand away from the thorax like wings. The distal parts of the extremities, particularly the hands and fingers, tend to remain free of trouble, but in spinal myopathia these are usually involved first and produce a claw hand. Corresponding to the various conditions of the musculature, in some parts of a muscle there is plain atrophy, in others an increase in volume and a change in the architecture depending upon the true and false hypertrophy, whereas in spinal myopathia the muscles are never hypertrophic. (Details by Pappenheimer.) The visible disappearance involves especially the muscles which fix the shoulder blade (pectoralis major, latissimus dorsi, anterior serratus), but also the biceps, the quadriceps femoris and others, while the increase in volume takes place in the calf muscles, the gluteal muscles, the infraspinatus, the deltoid, the triceps and others. The muscles of the face, *i.e.*, the orbicularis oris (the mouth remains slightly opened) and the orbicularis palpebrarum (closure of the lids is incomplete), may be involved. The face of myopathia is rigid and mask-like. The electrical irritability of the diseased muscles is quantitatively diminished. In contrast to spinal myopathia, reactions of degeneration are never found; fibrillary tremors never occur. Later, the tendon phenomena disappear.

Several varieties of primary myopathia may be recognized, although there are actually no essential differences: (*a*) pseudohypertrophy (lipomatosis luxurians progressiva), develops in earliest childhood, prefers the male sex, and involves chiefly the muscles of the calf, the thigh, the back

and pelvis. The change in the muscles continues, while their strength becomes less. The weak calf may become as thick as that of an athlete. Finally the muscles are transformed into yellow masses of fat. (*b*) The infantile form (Duchenne), is characterized by primary involvement of the face muscles, particularly the orbicularis oris and palpebrarum. In well marked cases, there is a mask-like, stupid face, the facies myopathica (Dejerine-Landouzy). (*c*) The juvenile form appears in youth and middle age and involves particularly the musculature of the shoulder girdle and the upper arm; the affection is usually confined to isolated muscles. (*d*) The hereditary form (Leyden) frequently begins in the eighth to the tenth year of life, or later, with weakness in the back and in the legs.

Comprehensive literature by Lorenz; see also Pappenheimer concerning juvenile familiar muscle atrophy.

Degenerations.—In cloudy swelling or acute granular degeneration, the cross striae are obscured by the granules of albumen which disappear after the addition of acetic acid. The change follows circulatory disturbances, in the neighborhood of inflammatory masses etc., and may proceed to fatty degeneration.

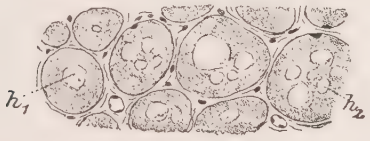


Fig. 989.—Cross section through muscle bundle with hydropic or vacuolar degeneration; h-1 and h-2, larger and smaller droplets of fluid in fibres. From a pale gelatinous muscle from calf of leg. (Med. power.)

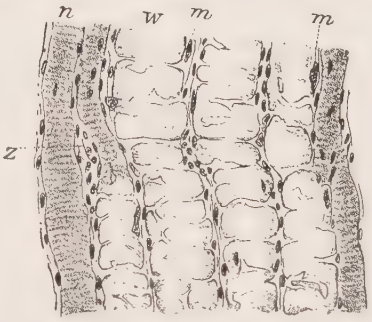


Fig. 990.—Waxy degeneration of rectus abdom. in typhoid; n, cross striated unchanged fibre; the lower part of adjoining fibre and three others completely glassy and waxy; m, proliferated sarcolemma nuclei; z, cellular infiltrated int. perimysium. (Med. power.)

In fatty degeneration, very fine fat droplets are seen in the contractile substance; these may, by confluence, become larger droplets. The cross striae are soon lost. This occurs in chronic cachexias, *e.g.*, phthisis, in many poisonings, *e.g.*, phosphorus, also in diphtheria (Hotzen).

Even forty-eight hours after poisoning with phosphorus the muscles may be found cloudy yellow red to yellow, soft and moist.

Regressive fat metamorphosis takes place in muscles whose function has been removed, thus in the deeper muscles of the sole in flat foot (A. Hofmann). In deciding upon the importance of fat changes in the muscles, it must be remembered that fat in varying amounts appears normally in the muscles, richly in the diaphragm (Wegelin), and that this fat may be increased by a fatty diet, that it disappears in starvation, and that morphologically it cannot be distinguished from the fat produced by

pathologic conditions (Surbek). See also Kolodny concerning fatty changes in the voluntary muscles. In hydropic or vacuolar degeneration, clear droplets are found microscopically in the muscle fibers which grossly are watery and very pale (fig. 989). In fibrillar segmentation, there is solution of the substance of the muscles. This is seen, *e.g.*, in edema of the extremities of some duration. In laminated destruction of the sarcoplasm, all kinds of fragments are found.

According to Grawitz, (see Landois), fibrillar segmentation of contractile substance makes it change to connective tissue, a myogenous connective tissue.

Waxy or hyalin degeneration, (fig. 990), was described first by Zenker in typhoid fever, in which it involves chiefly the abdominal muscles, particularly the rectus, and the adductors of the thigh, but may also appear in those of the tongue. It consists of a homogeneous glassy change of the cross striated sarcolemma content, so that this substance swells and breaks up into small segments corresponding to the cross striae. The hyalin content of the sarcolemma then disappears into smaller and smaller masses, which become absorbed. This hyalin change is also called coagulation necrosis.

According to Thoma, it is caused by self tearing, and is a hyalin swelling and infiltration of leucocytes in the contractile substance with loss of the doubly refractive granules; it precedes liquefaction and absorption (for other views, see Beneke).

According to Beneke, quite similar changes caused by swelling and coagulation of the muscle substances occur in smooth muscle.

If the condition is extensive, the muscles become cloudy, pale, fleshy, waxy and lose their rigidity; they tear easily in the presence of subfascial or intramuscular hemorrhages which may be quite extensive. Such hematomas in the muscle are relatively frequent in the rectus abdominis in typhoid fever (fig. 988).

The etiology of the change is quite varied. It is seen particularly in typhoid fever and in paratyphoid B. (Sternberg), but also in other infectious diseases such as tetanus (particularly in the thorax, Aschoff), tuberculosis, variola, recurrent fever, influenza (Zausch, Kuczynski-Wolff, lit.) and others; furthermore, it is seen in poisonings from snakes and other intoxications, as well as in anaphylactic shock, in which there is serious waxy degeneration of the diaphragm (Beneke), after burns and frost bites, following traumata (self tears, electrical shocks), (M. B. Schmidt), in trichinosis, in which hyalin degeneration follows the action of the poison on the fibers which do not contain the trichinae, and also following the stasis in icterus neonatorum (Heydrich). Those muscles are particularly involved which are subject to stretching and much use;

it is thought that these conditions favor the lodgement and activities of the poison (Zausch, lit.). Severe muscle necroses, particularly in children, have been observed after the injection of salvarsan; sepsis may follow the condition.

Those degenerations mentioned above are met under various conditions; thus after overexertion, crushes, tears, lowered nutrition from general or local causes (decubitus, hemorrhages), inflammations, infections and intoxications.

Following the various forms of simple and degenerative atrophy which cannot always be sharply separated (Rosin, lit.), there occurs regenerative growth of the muscle nuclei (fig. 990); these are gathered in rows and masses, and may be retained long after the disappearance of the muscle fibers—or they may be established as uni- or multinucleated sarcoblasts on the outer side of the destroyed content of the sarcolemma. There may be more or less complete replacement, (more details in section following); in typhoid fever it is almost complete, and connective tissue scars appear only where larger hemorrhages and ruptures took place or where there was extensive necrosis. Marked fatty changes and the appearance of regenerated muscle cell tubes in poisoning by agaricus phalloides is of diagnostic importance in relation to phosphorus poisoning and acute yellow atrophy of the liver (p. 910).

See Schmincke (lit.) concerning gunshot wounds of the muscles. Among the injuries due to blunt force, those necroses from concussion have awakened great interest (Borst, Kuttner); without an external wound, there is extensive necrosis of large areas of muscle tissue, which take on a gray yellow, dry, fish flesh character. There may be no hemorrhages in the tissues. Ischemia following spasm or paralysis of the vessels with stasis are held responsible for the necroses. Neither severe lesions of the vessels nor thrombi can be found.

Amyloid change of the internal perimysium and of the sarcolemma is very rare. It has been observed by Ziegler and others as nodules in the muscles of the tongue and larynx, and the author saw it occur in the smooth musculature (Johanni, Hueter).

Calcification takes place in calloused muscles, in local necrotic muscle fibers (Pielsticker, lit.), in the neighborhood of tuberculous abscesses, etc. A generalized deposit of calcium (calcinosis) in the subcutaneous and intermuscular tissue following trauma or a previous rheumatism is quite rare (Lhermitte).

Inflammation of the Muscles (Myositis)

Myositis follows by direct extension from the neighborhood, *e.g.*- from a phlegmon of the skin and fascia, leg ulcer, decubitus, etc., or trau,

matically following crush wounds, tears, or from the lodgement of inflammatory organisms from the blood stream.

Milder forms of inflammation show serous saturation and cellular infiltration of the perimysium; the muscle fibers themselves remain intact or become cloudy, swollen, show fatty degeneration or coagulation necrosis.

Myositis with sero-cellular exudation occasionally follows injury to muscles, anthrax extending from the skin, or is the initial stage of a severe myositis. In trichinosis, round cell infiltrates appear in the interstitial tissue independent of the parasites. In many cases the degeneration of the contractile substance is more marked than the inflammation in the interstitial tissue. The partly degenerated fibers may later be replaced by regenerative growth. In rheumatic myositis there are nodules in the muscles similar to those in the myocardium (p. 63; Grauhan).

In the rare multiple muscle inflammations—acute or chronic polymyositis (E. Wagner)—the muscles of the extremities, the buttocks, and the surrounding soft parts swell acutely and give the symptoms of pain and disturbance in the general well being. The disease is accompanied by fever and is probably of infectious origin (Mayesima); it has been observed following influenza, angina, articular rheumatism, meningococcal infection, suppurative processes such as tonsillar abscesses, etc., and also in epidemic form. Landsteiner found a streptococcus, identified by v. Wiesner as the streptococcus polymorphous, which, according to him, is also the cause of lethargic encephalitis (p. 1900). Edema of the skin and the subcutaneous tissue may be marked enough to obscure the swelling of the muscles. The musculature appears decolorized, pale, like fish flesh, and is granular, waxy, or vacuolar; the number of nuclei is increased; in the interstitial tissue there are edematous swelling, collections of leucocytes and lymphocytes, small and larger hemorrhages; the degree of this interstitial change is variable. Soon the muscles, very tender to pressure, are unable to function and the patient lies helpless. The trunk muscles are most frequently diseased and the respiratory or deglutition muscles but rarely. Lighter cases end favorably. Severe cases end in weeks or months by death from asphyxia or aspiration pneumonia. The disease resembles clinical trichinosis and was called pseudotrichinosis by Hepp. Since the edematous skin is usually red, and sometimes shows exanthemata, Unverricht calls the condition dermatomyositis (see also Henschen, Lohlein and Schlossberger). Chronic cases may last one to two years.

See Schwarz concerning the tuberculous form of polymyositis, and for the frequent occurrence of polymyositis in tuberculous individuals, see Kankeleit (lit.).

Suppurative myositis is first an interstitial inflammation, which appears in foci if of hematogenous origin; when occurring by extension from the neighborhood, it is diffusely spread and leads to circumscribed abscesses, or diffuse phlegmons in the interstitial tissue. The muscle substance itself undergoes degeneration or necrosis over larger or smaller areas. Small, usually multiple, abscesses, such as, for example, in pyemia, appear as sharply circumscribed cavities filled with pus and debris, while abscesses which form in the neighborhood of a phlegmonous myositis are

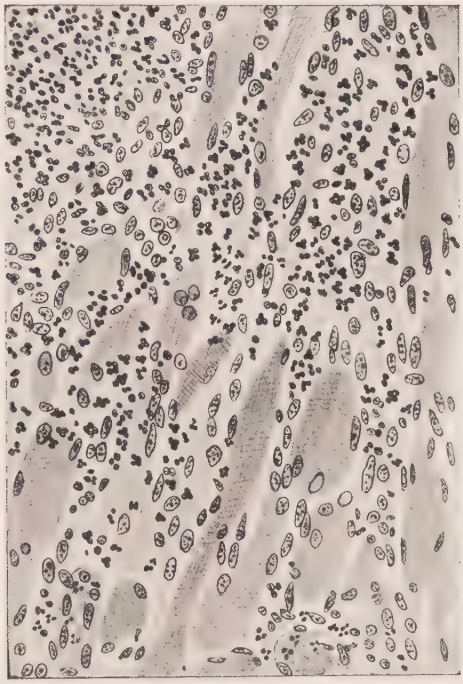
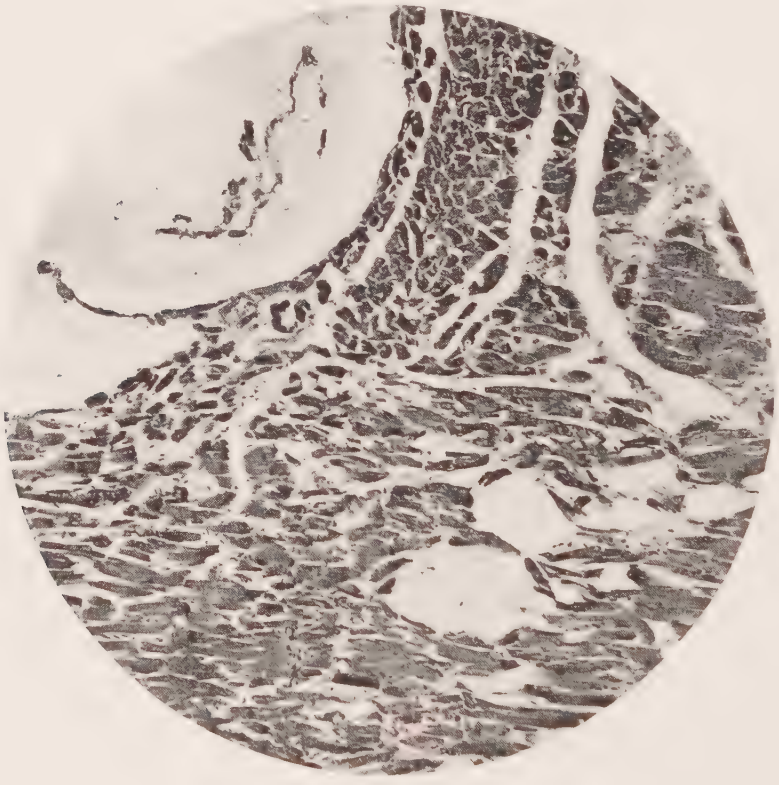


FIG. 991.—Suppurative myositis (abscess formation). Besides the leucocytes, which completely destroy the muscle fibres, numerous proliferated muscle nuclei are seen; man 24 years old. (High power.)

less sharply bordered, and blend into the softened, purulent neighborhood. Phlegmonous myositis may also have an ichorous character, and the necrosis gives the musculature a discolored black brown or black gray or gray green pulpy or smeary appearance. This is seen particularly often in fecal abscesses, but also in septic wounds and in sacral decubitus; in the latter, the muscle fibers become dry and brittle. Abscesses may be encapsulated by connective tissue proliferation in the neighborhood, and after emptying, are transformed into a scar. Quite regularly, there is marked growth of granulation tissue with its outcome of scar formation in the chronic muscle suppurations of tuberculosis, actinomycosis, glanders, etc.

According to Saltykow, the pus in suppurative myositis consists, not only of leucocytes, but also of growing muscle and tissue cells, which, according to Busse, contradicts the older viewpoint of Grawitz and his scholar, Krosing, of the active participation of the tissue in the inflammatory process.

In the **gas phlegmons** or gas edema seen so frequently in the war, the musculature is much involved (typical site, Franz). This condition,

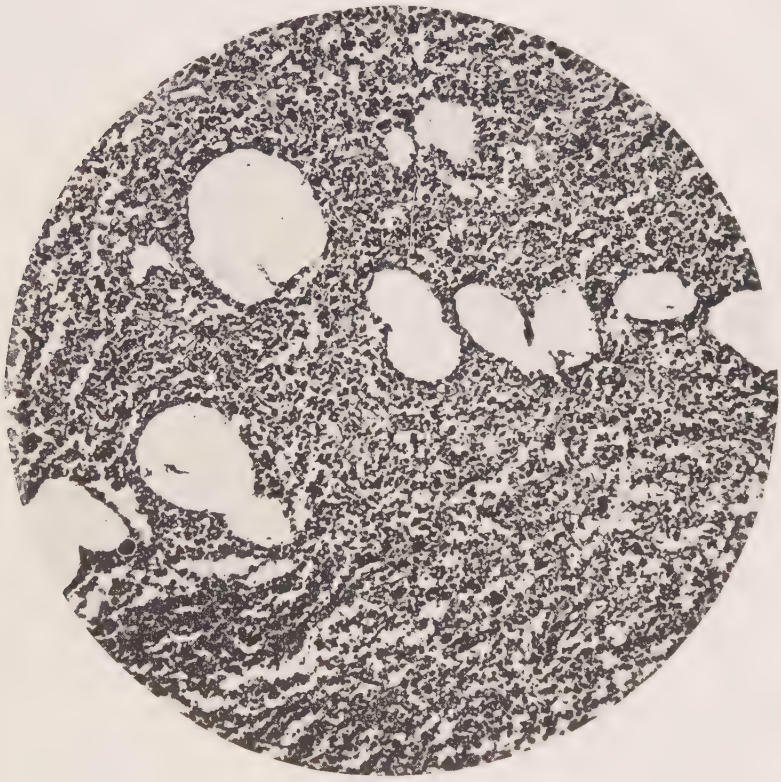


Heart, gas infection

FIG. 992.—Sections of myocardium spleen kidney and liver from a case of gas bacillus infection. This patient, man 30 years old, had a swelling in the middle of his right tibia which was incised as an abscess. A small amount of pus was obtained and considerable friable, granular, blood stained tissue, which proved to be spindle cell sarcoma. The patient died four days later of general gas bacillus infection. The gas filled spaces are plainly discernable in the sections. All of the organs grossly were swollen, soft, pale, spongy and honey-combed with innumerable tiny cavities. × 100. L. H. #256.

caused by the bacillus phlegmones emphysematosae (Fraenkel; pp. 1755 and 2070), consists of a particular form of death of the connective tissue and the musculature without microscopic pus formation. The muscles are saturated with gas bubbles (CO_2 from the muscle glycogen, see Pribram), become discolored, at first resemble a dried salmon, then become necrotic, soft, vacuolar and waxy with laminated destruction, then soft

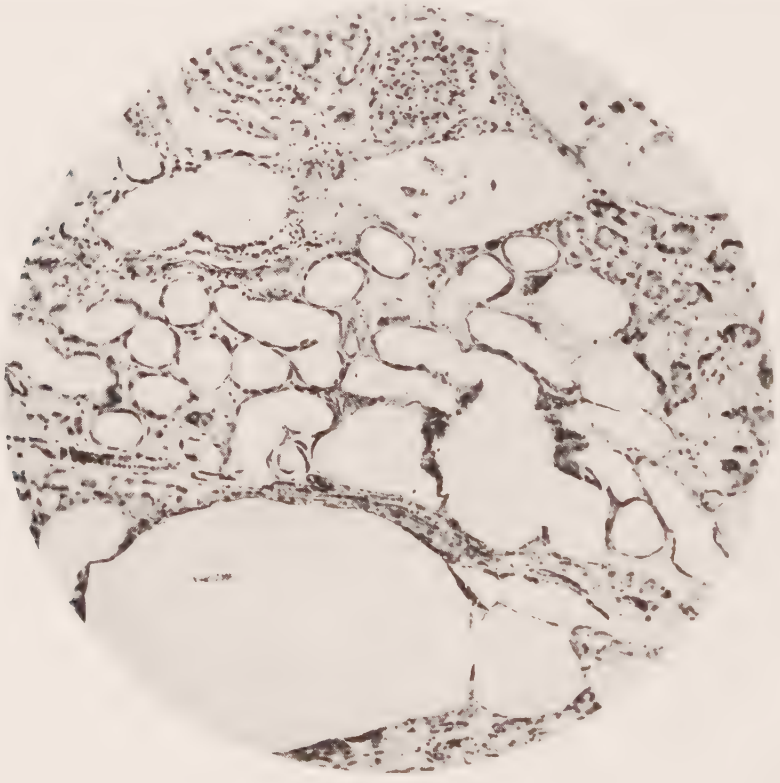
and fluid, until finally they become a pulp filled with gas bubbles. There may be combined with this an edema of varying degree in the interstitial tissue which becomes cloudy and, following hemolysis, red in color; microscopically there are innumerable bacilli, fibrin, and leucocytes in the edematous mass. When a thigh is the site of gas edema, the swollen, pale skin feels cold to the touch (Heyde), and if it is cut through, the musculature crepitates. This is a decomposition necrosis (Hitschmann



Spleen, gas infection
FIG. 992.—(*Continued.*)

and Lindenthal); the gases (CO_2) arise from the action of the gas bacilli on the necrotic tissue. In addition to the immotile gas bacillus which is by far the most important organism, another motile anerobic bacillus of Ghon-Sachs may be met, which likewise belongs to the type of lactic acid bacilli, and which is, according to the description of v. Baumgarten, similar to the organism described by Aschoff, Conradi and Bieling, or at least, is quite closely related. This latter question cannot be discussed here. Death occurs from toxemia (there is no swelling of the spleen); there do occur cases, however, which recover.

In addition to these pure cases, there are others due to mixed infection; since splinters of shell often carry into the tissues pieces of earth infected with various anerobic organisms and spores of many other kinds of bacteria, it is not surprising, as v. Baumgarten remarks, that a mixed infection appears, in which pathogenic anerobes of various kinds mingle with saprophytic decomposition organisms and pyogenic bacteria. This accounts for the varying pictures: that of a markedly phlegmonous suppu-



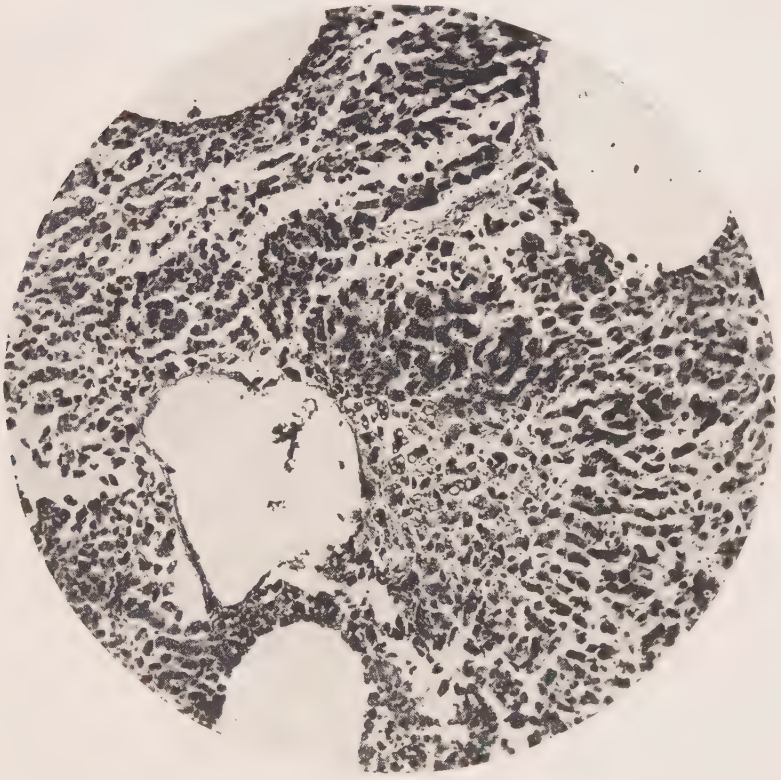
Kidney, gas infection
FIG. 992.—(*Continued.*)

rative process, with or without decomposition, or a predominant edema, or a non-putrid necrosis with gas formation without odor, or foul decomposition with foul smelling gas formation, etc. The differences are expressed in the nomenclature so frequently used, such as gas edema, gas phlegmons, gas gangrene, etc. It is important to remember in the complicated bacterial findings that the gas bacillus of Eug. Fraenkel is the principal invader, and the principal pathogenic organism producing gas; the bacillus of malignant edema (p. 2070) represents a fundamentally different group (Ghon and others). See also Heyde, Stemmler, Weinberg and

Seguin, Coenen, (lit.), Aschoff, (lit.), and Schmincke, war diseases of the cross striated musculature.

By **myositis fibrosa** (fibroplastica) is understood a productive connective tissue development in the muscles, in which the muscle fibers undergo destruction to various degrees.

It is almost always secondary to other conditions, thus, as a result of acute or chronic degenerative and inflammatory processes in the muscles



Liver, gas infection
FIG. 992.—(*Continued.*)

themselves. The milder degrees follow, *e.g.*, a spinal and primary muscular atrophy; more severe forms are seen in the neighborhood of chronically diseased bones and joints, in chronic conditions such as eczema, leg ulcer, etc. The muscles in the beginning are marbled by white fibrous strands and septa and are later totally replaced by connective tissue. When a muscle abscess is cicatrized, it occurs by way of a myositis fibroplastica.

The condition known as caput obstipum musculare, or wry neck—Kader does not believe that the condition is congenital, *i.e.*, intrauterine—is brought about by injury at birth to the sterno-cleido-mastoid muscles,

in other cases by a hematogenous infection, but in most, by a coincident activity of both of these factors. A rich connective tissue develops at the cost of the disappearing muscle fibers. This opinion is substantiated by Hildebrand, as well as by the experiments of Heller. According to Couvelaire, the hemorrhages, sometimes considered traumatic (p. 2034), are dependent upon the myositis (Durante). Kempf, on the other hand, believes they are of ischemic origin, and Schloessmann (lit.) shares this opinion; the causes of the ischemic muscle paralysis and the contracture are intrauterine difficulties from lack of space. According to Sippel, who studied intrauterine pressure by means of the x-ray, hemorrhages precede the fibrous changes in the muscle, while Schubert (lit.) disagrees with the intrauterine pressure theory and believes there is a primary hereditary central nervous disturbance (see Gauss); see Henschen concerning wry neck in dyspnoeic goiter patients.

Infectious Granulation Tumors

Tuberculosis of the muscles is fairly uncommon. The condition may extend directly from tuberculous bones or joints, or from a tuberculous pleuritis into the intercostal muscles. This may lead to cicatrizing thickenings, or caseous softened tuberculous nodules or fistulous abscesses, occasionally sunken below the surface (see under spondylitis tuberculosa, p. 1127). Tuberculosis may also reach the muscles from the mucous membrane (tongue tuberculosis, p. 555) or from the external skin (lupus).

Hematogenous muscle tuberculosis is rare; it occurs in general miliary tuberculosis, or is independent of this condition; in the latter case isolated muscles show diffuse tumor-like tuberculous infiltrates, or tuberculous invasion characterized by miliary nodules and accompanied by granular and fatty degeneration of the muscle substance and hemorrhages. The surgeons call such cases "primary" tuberculosis of the muscles, *i.e.*, a lesion not spread from the neighborhood (Zeller, Kaiser). Pilliet discusses the histological details; Saltykow (lit.) showed experimentally that even in the first days of the small cell infiltration, as well as in the formation of the epithelioid cells, the muscle elements take active part, a fact which was pointed out before by Krosing (Grawitz; see also Hungerbuhler).

Syphilis of the muscle occurs (1) in the form of a chronic cicatrizing thickening of the muscle connective tissue, which leads to atrophy of the muscle parenchyma (syphilitic fibrous myositis). By preference it involves the biceps and occasionally the masseter muscle, and often leads to contracture. Syphilis also appears in the form of gummata which spread into the neighborhood, so that one in the region of the skin appears as an immobile protruding tumor, with a tendency to destruction and of a size larger than that of gummata in other situations. They may pro-

duce deep crateriform ulcers, which usually heal with deep scars. Interstitial myositis is combined with gummata so that the latter seems a caseous dry mass embedded in connective tissue.

The disease appears in various muscles such as those of the upper extremities, particularly the biceps, but also in the sterno-cleido-mastoid, muscles of the back, in the tongue and in the muscles of the eye (Busse). The preparation illustrated in fig. 993 is from the back of a young woman who had an ulcerating crateriform gumma of the skin and musculature as large as a fist; this unusually large syphilitic growth was partly softened and destroyed, partly fleshy, and partly caseous and dry.

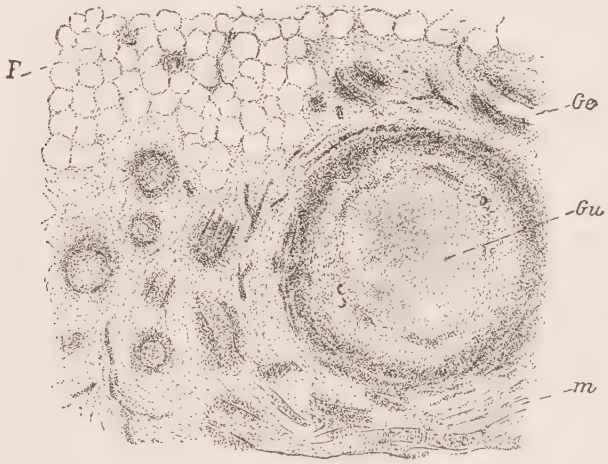


Fig. 993.—Gumma, rounded, centre caseous, lighter spindle cell zone contains three giant cells (Langhan's type); broader dark outer zone of granulation tissue. In the surrounding cicatricial tissue still occasional muscle fibres (m). Vessels (Ge) chiefly veins, changed by vasculitis and perivasculitis, partly obliterated, and surrounded and infiltrated by numerous round cells. F, masses of fatty tissue. The section comes from the margin of the large disintegrated gumma of skin and musculature of back, mentioned in text. (Medium power.)

General Remarks Concerning the Microscopic Structure of a Gumma. The Differential Diagnosis, Particularly from Tuberculosis

Microscopically, small gummata give the appearance of clover leaf or rounded structures (fig. 993). In general, the syphilitic connective tissue undergoing caseation, in contrast to tuberculous tissue, has much more tendency to diffuse spread than to the formation of nodules (Mandl). In the beginning, it is composed only of richly cellular granulations or of predominant epithelioid structures with few giant cells in the centers (see also Schellhorn); gummas of the cross striated musculature are exceptions, since giant cells are always present (see Landois); the periphery is composed of round cells, as are found occasionally in the smallest so-called miliary gumma, *e.g.*, in the skin. But in contrast to most older tubercles (p. 1493), these structures retain numerous blood vessels, a fact which v.

Baumgarten believes is quite decisive, whereas caseation may be entirely lacking. Somewhat older, well formed, gummas usually show three zones; (*a*) a caseous coagulation necrotic center, which, however, even in older gummas, often retains the fibrous character of the interstitial substance, and allows the demonstration of elastic fibers and the contours of vessels after proper staining; external to this there is (*b*) a middle zone of loose spindle cells (fibroblasts) with epithelioid cells, round cells and usually isolated (with the exception of muscle gumma) giant cells, quite similar to those found in tuberculosis (Wakabayashi), all in a richly fibrous connective tissue; external to this there is (*c*) an outer zone of younger granulation tissue rich in round cells (lymphocytes, plasma cells, few leucocytes) and vessels. These zones must be considered as a growth directed from the periphery of the connective tissue (outer zone), and the changing of this zone to scar tissue (middle zone) which the caseous gummatous growth partly invades, partly goes through, and partly surrounds as parts of it are absorbed. This outspoken tendency to fibrous change even in extensive gummas, differentiates the syphiloma from the tubercle. In contrast to a tubercle, there is a much less sharp boundary between the gumma and its surroundings. Thus gummata often lie in cicatrizing connective tissue. The vessels (elastic staining) which pass through and often remain in the middle of the caseous material are diseased, particularly the veins (Rieder); all coats may be infiltrated by cells and thus thickened (vasculitis), and also the neighborhood of the vessels is infiltrated by round cells and plasma cells (perivasculitis); the lumena may be narrowed or even entirely obstructed by growth of the intima. Vessel branches which are retained long in the gummatous growth produce the above mentioned clover leaf shapes. The contour of the tissues, or the architecture disappears, but more slowly in gummatous masses than in those due to tuberculosis. In their centers, blood filled vessels are often found and a fibrous appearance or shadow pictures of the original structures may be identified even in old dried caseous masses. This is a most important differential point from tuberculosis. In syphilitic granulation tissue, Rossi occasionally found numerous eosinophilic cells in contrast to the few in tuberculosis and the numerous cells of this kind in actinomycosis. An examination of caseous gummas for spirochetes practically always results in negative findings (p. 1918).

The differential diagnosis (see also pp. 1493 and 1918), in doubtful cases, particularly in very small gummata, may be facilitated by staining for tubercle bacilli. It is also necessary to distinguish the tissue from that of sarcoma. See p. 1493 for the details of elastic tissue staining.

In **glanders,** abscesses, as well as suppurative liquefaction of large numbers of granulation nodules, may appear in the muscles and between

them. Glanders (p. 298) may be spread through the lymph or blood channels (see under skin).

In **actinomycosis,** the granulation tissue has a tendency to fatty changes (sulphur yellow color), cicatrization, the formation of fistulae and abscesses. As a rule, the infection occurs by continuity from a neighboring part, thus, for example, from actinomycosis of the pleura (p. 466).

Lipomatosis. True Hypertrophy. Congenital Myotonia

Lipomatosis.—As a part of the picture of various kinds of atrophy, the internal perimysium becomes markedly developed and more richly nucleated than normally; indeed it is changed frequently into fatty tissue. Thus the atrophy of the muscles may be masked. This is seen, for example, in the spinal paralysis of children (p. 1990). Lipomatosis in progressive muscle atrophy may even lead to an increase in the volume, and thus the condition is spoken of as lipomatous pseudo-hypertrophy (p. 2036).

True hypertrophy of the muscle fibers depends upon thickening and elongation (Morpurgo, lit.), and perhaps also on increase in their number. Increase of work may lead to functional or activity hypertrophy.

Schiefferdecker assumes that the composition of the sarcoplasm is changed in the hypertrophy of activity because the number of nuclei is less in contrast to the ordinary fibers (see p. 2032); the length of the nuclei remains the same, their thickness increases, so that the relative mass of nucleus becomes diminished.

Uncommonly, a true hypertrophy occurs without increased work, as for example, after typhoid fever. In progressive muscular dystrophy (p. 2035), true hypertrophy may also occur. The same is assumed to occur in Thomsen's disease or congenital myotonia.

In Thomsen's disease, Erb, Dejerine and Sottas, Fulda and others excised small pieces of muscle and found a thickening of the primitive fibers to double their size, increase in the number of nuclei, some obscurity of the cross striae, a homogeneous appearance of the cross section, and other such conditions. M. Heidenhain records the interesting finding, in cases of longer duration, of queer cross striated fibrillae in the hypertrophic muscle fibers, which were differentiated from the external layer of sarcoplasm as bundles surrounding or passing through the isolated fibers circularly or in cork-screw manner and in layers of varying thickness; these "rings," he believed to be antagonists of the longitudinal fibrillae, developed for the purpose of neutralizing the increased size of the cross section of the muscle fibers, so that they could not contract over-vigorously. The disease usually attacks several members of a family, and appears in a number of generations; thus, for example, in Thomsen's own family, it

appeared in four generations. Beginning in early childhood, it manifests itself as a hypervoluminous condition of the musculature, with diminished strength, in stiffness of the muscles, and transient cramp-like contractures when a muscle is voluntarily brought into action after it has rested for a longer time; thus the out-stretched arm may suddenly become stiff. On shaking hands, the individual cannot quickly release the contracted muscles. Voluntary movements, fatigue, etc. increase the myotonia. The entire musculature may be involved, but in varying degrees. In the muscles the objective findings, as Erb showed, are (*a*) increase of the mechanical muscle irritability,—tapping of the muscles calls forth tonic contractions of the touched part with prolongation of the contraction. (*b*) Changes in the electrical irritability,—the myotonic reaction (Erb). Of particular interest is: on application of strong galvanic currents, "consecutive rhythmic wave-like contractions run over the muscles, which, as though obeying a law, come from the cathode, and spread to the anode."

Myotonia atrophicans (see also Naegeli) is probably related fundamentally to Thomsen's disease (v. Bezold, lit.); this condition is called myotonia dystrophy by Hauptmann; paretic, atrophic and myotonic symptoms appear simultaneously, or the atrophic changes precede those of myotonia; the active myotonic symptoms are localized to the closing of the fists; dystrophic changes are found mostly in the face, muscles of mastication, the sterno-cleido-mastoids, the forearm, the peronei. In addition, there are symptoms of posterior column disease, sexual impotence (atrophy of the testes), baldness, cataract, etc. According to Hauptmann, internal secretory factors are important. See also under myasthenia gravis, p. 1995.

Regeneration of Muscles.—The way in which regeneration takes place depends principally upon the kind of damage done. Complete regeneration occurs (Volkmann), when the damage involves only the contractile substance and spares the protoplasm, sarcolemma and the muscle nuclei, as in typhoid fever, frost bites, occasionally in crush wounds, (Pielsticker). The destroyed parts of the fibers are replaced by a new formation which begins in the nuclei still present in the tube and in the sarcoplasm; it follows along the lines of the embryonal types of muscle fiber formation. Traumatic injuries are followed by restoration of muscular continuity only when there is a small amount of tissue lost, *e.g.*, total section; otherwise union takes place by connective tissue; later, a matter of weeks, new muscle fibers grow into the scar from muscle buds of the old fibers.

Regeneration begins in the nuclei of the old fibers by an increase in their number, partly by direct division and partly by mitosis in the living muscle fibers between fresh spaces, *i.e.*, from pieces which do not touch,

but are not widely separated. New sarcoplasm is then produced. According to the experiments of Thoma, the nuclei "flow" into the region of the injury. New fibers then appear, first in these richly nucleated masses of sarcoplasm; the fibers are in continuity and their ends grow in length and thickness and form the so-called muscle buds (Neumann, Nauwerck), into which the still retained cross striated substance enters. Fibrillae are then differentiated in the muscle buds; the longitudinal striae appear first and then the cross striations. In addition, before the formation of the buds or afterward, some outgrowing muscle fibers may split longitudinally into two or more young fibers. If a space is present between severed ends, buds appear on each side; if the sarcolemma is cut across, terminal muscle buds are formed. Nuclei with no connections with living muscle fibers soon look like large epithelial cells, and after repeated division become multinucleated masses of protoplasm (Waldeyer), most of which are destroyed by way of fatty degeneration (Nauwerck). Some few become sarcoblasts, developing into independent muscle fibers (of embryonal type); perhaps these discontinuous muscle cells are finally joined to old fibers or to muscle buds, but some become giant cells which remove remnants of the protoplasmic substance, or at least, surround them. Section of the nerve is without influence on the course of regeneration (Kirby), but intensive inflammations so interfere with nutrition, that regeneration can not take place.

Lit. concerning muscle regeneration by Pielsticker, Landois; concerning regeneration of transplantation by Goldzieher and Makai; Wullstein.

See Berry (lit.) concerning regeneration and regenerative new formation of smooth muscle fibers and cells.

Myositis Ossificans

Intra- and Intermuscular Formation of Bone.— Under various pathological conditions, bone is developed in the connective tissue in and between the muscles. The process is like periosteal or intramembranous bone formation (p. 1061), *i.e.*, the bone, or rather its osteoid prestage, develops: (*a*) directly from germinal tissue (chiefly growing connective tissue,—cell metaplasia), or from mature connective tissue, *i.e.*, cells and intercellular substance (tissue metaplasia), or (*b*) indirectly, *i.e.*, with an intermediate stage as cartilage—cellular or tissue metaplasia or enchondral ossification, *i.e.*, solution of the calcified cartilage and the formation of marrow spaces by young cells. Calcified connective tissue is occasionally changed into bone by the same means. Trabeculae of bone, once formed, grow via the activity of osteoblasts, which, generalizing, do not necessarily come from periosteum, but may be descendents of connective tissue cells (Kawashima).

The term "osteoma" is used, but this condition has nothing to do with true "osteomas"—true tumors. The term "inflammatory osteoma formation" is also used.

According to Lexer, proliferation of connective tissue cells gives rise to a germinal tissue with new fibrillae which change into cartilage and then into bone. We might speak therefore of the stage of connective tissue induration in myositis ossificans.

The disease is also called myositis interstitialis ossificans (Munchmeyer), for bone is established not only in the soft connective tissue between the isolated muscles, and between individual muscle bundles, but also in the tough connective tissues of the fascias, of the tendons, the intermuscular fascial sheaths and the ligaments (Mays). The muscle fibers remain quite passive, but are compromised and undergo simple atrophy; they occasionally show growth of nuclei, but this has no significance in the process.

The disease occurs in two forms known as the circumscribed and the multiplex, or as the stationary and the progressive (Cahen). It is not good terminology to speak of the condition as "osteomas," "muscle osteomas," etc. Comprehensive literature by Kuttner; see also Manuwald.

Myositis Ossificans Circumscripta.— Bone formation begins in isolated muscles subjected to a single o repeated trauma or influenced by chronic inflammations in neighboring bones, *e.g.*, the hip joint in tabes. As typical examples are mentioned: localized bone formations in the arm in luxation of the elbow (Machol); in the deltoid and the pectoralis muscles, as the so-called bayonettier's bones from the impact of the gun, etc., (Busse and Blecher, Kawashima), in the biceps of gymnasts, the adductors of the upper thigh or of the vasti in horseback riders, from single massive tearing or repeated smaller tears (Knaak), etc. The bones are shaped like cones or thorns or plough-shares; some are connected to the main bone as immovable exostoses pushed between the muscles, fascias, tendons, etc., as continuous fascial, tendonous, ligamentous, or intramuscular osteomas. According to Sudeck, the process begins in bits of periosteum, or in transplants. According to Busse, hemorrhage is important in traumatic myositis because it crushes the muscle fibers and stimulates connective tissue production (Kawashima), with subsequent ossification. Thus, larger intramuscular hematomas or cysts, may have bony walls and fluid contents. Berndt believes, however, that it is necessary to add a mild hematogenous inflammation (Ropke, lit.). Traumatic myositis ossificans has a tendency to spontaneous cure. (See also Godler, Mackins, Fabris, Gruber, lit., Krieg.)

The other form, *i.e.*, myositis ossificans progressiva or multiplex, begins spontaneously, usually early in youth, as a rule, first in the muscles

of the shoulder, nape of neck, the masseters, and then gradually spreads to the muscles of the back until all the muscles are involved. In some cases, malformations of the extremities, particularly microdactylia, have been observed. Usually the disease begins as the doughy, painful swelling of an infiltration (first stage), and is followed by fibrous induration (second stage), from which directly or indirectly, according to whether cartilage is formed in the second stage or not, the bone is developed (third stage), (Munchmeyer, Lexer). All three stages may be present at the same time in different muscles of the same individual. The muscles themselves

FIG. 994.—Large antler-like exostoses of hyperostotic right femur about at the insertion of vastus int. and adductor magnus. In the muscle itself in the long axis to the bone was a stylus-shaped and apparently broken off, tendonous bone.

FIG. 995.—Pronged inflammatory osteoma of ileopsoas muscle in psoas abscess and caries of lumbar spine and os sacrum. There was also an exostosis on the ilium. Woman 37 years old; 9/10 nat. size.

remain quite passive; some undergo destruction because of pressure, others because of inactivity (Cornil and Coudray). Goto (lit.) calls the condition "hyperplasia fascialis ossificans progressiva." The newly formed

bones acquire various shapes. Along the back, they are flat, angular plaques; the arches and spinous processes of the vertebrae may be joined by bridges, as in the case studied by v. Recklinghausen, the skeleton of which is preserved in Strassburg. In the long muscles such as the flexors of the upper arm, the bones are cylindrical in shape and force the extremities into angular contractures. Bridges of bone in the masseter muscle fix the lower jaw. In other situations, they are angular, nodular, or antler-like, or thin, long, lead-pencil shaped, fixed to the periosteum as exostoses, or as discontinuous masses spreading into the soft parts. As a rule, the combination of multiple exostoses and muscle osteomas is quite frequent, without the direction of ossification necessarily determined by muscle pull. If the joints are ankylosed, usually in angular positions, the "ossified" patient becomes "brittle" and stony.

Etiologically, very little is known of myositis ossificans progressiva, (lit. by DeWitt). Relationship to diseases of the spinal cord (Eichhorst) and to syphilis (Lang) has been postulated. To explain the bone formation, particularly that in the muscles, it has been assumed that the periosteal osteogenetic layer of the corresponding bone takes some part. If the intermuscular connective tissue is looked upon as belonging to the skeleton of the locomotor apparatus (Koester), we may, as v. Recklinghausen emphasizes in the sense of Mays and others, think of a congenital diathesis toward the formation of bone, so that a trauma during birth or later becomes a minor cause; the boundary between the periosteum and the intermuscular connective tissue is removed, or perhaps shoved peripherally between the muscles, and the intermuscular connective tissue takes on a periosteal character. This hypothesis seems more plausible than that which assumes that osteoblasts wander in from the periosteum, or have been misplaced during fetal life. According to Virchow, the conception of the "ossifying diathesis" must be extended and not confined to certain preexisting structures, since ossification may be spread diffusely over situations such as subcutaneous tissue, which have nothing to do with bones and muscles. Recently, the congenital diathesis idea has been formulated thus: there is a congenital, non-hereditary, reduced ability of differentiation of the connective tissue elements from a disturbance in the mesenchyme cells (Stempel, Jungling, Frattin, Blenkle, lit.); as accompanying symptoms of the developmental inhibition there appear microdactylia and other malformations.

Relationships to diseases of the spinal cord, especially trophic influences, were postulated from the circumstance that tabes and syringomyelia are sometimes accompanied by ossification of muscles and the formation of exostoses in numerous situations. Thus the author saw elongated, pencil-shaped, movable osteomas in the musculature of the thigh of a tabetic

with a fracture of the neck of the femur, and similar conditions in cases showing tabetic arthropathia of the hip joint. A neurotic form of circumscribed myositis ossificans is also mentioned.

True Tumors of the Muscles

Primary new growths are uncommon. They begin most frequently in the muscle connective tissue. There occur: fibromas, usually the hard varieties, tumors seen particularly often in the fascia of the rectus abdominis muscle. Lipomas arise from the interstitial connective tissue or from the intermuscular tissues; the latter are rarer, they perforate the fascia, and then their chief mass is subcutaneous, whereas the pedicle is intermuscular. The thigh and the back are preferred. Myxomas usually have a colloid appearance; some are myxomatous sarcomas; sites of preference are the thigh, with an intermuscular position. There may also be mentioned chondromas (Honsell and others), the rarer chondrolipomas (Kolaczek), hemangiomas (Muscatello, Pupovac, Sutter, Kolaczek and others, lit. to 1906 by Putti and Reclus and Magitot, Finsterer, lit.). The author saw a cavernous angioma in the cervical musculature of a ten year old child, and in the quadriceps of an eighteen year old boy; hematolymphangiomas (Ritschl), cavernous angiolipomas occur; as an example, the author saw a large diffuse angiolipoma in the cheek musculature (Szenes, lit.). Osteomas have been mentioned; they usually belong to myositis ossificans. Sarcomas are not altogether rare. They may arise intermuscularly. Various kinds have been described; thus, fibrosarcomas, round, spindle cell sarcomas; the author saw a tough, rounded, closely applied, glassy, gray red, vascular, spindle cell sarcoma as large as an apple in the pectoralis major muscle of a forty-two year old woman, and a very polymorphospindle cellular sarcoma with giant cells in the biceps of a forty-one year old man; prolongations of the tumor invaded between the interstitial tissue into the axilla. Furthermore, there occur, myxosarcomas, angiosarcomas, giant cell sarcomas, one of which was seen by the author in the brachial muscle of a railroad worker; Ulrich described a malignant angioendothelioma, as well as one with cartilage and bone tissue (lit. by Landois). Rhabdomyomas can be found in the heart muscle, but are quite rare in the voluntary musculature; some were rhabdomyosarcomas or malignant rhabdomyomas (adults in the cases of Lambl, Buhl and particularly Fujinami; in a $3\frac{3}{4}$ year old boy in the case of Ritter, lit.; in a 7 months old child in the case of Warnery, lit.). See Busse, Kuttner-Landois, Landois concerning the tumors of cross striated muscles.

Among the **secondary tumors,** carcinomas are the most frequent; they usually appear by direct extension, *e.g.*, carcinoma of the breast into the pectoralis muscle, from a uterine carcinoma into the ileo-psoas

muscle, etc.; in a 45 year old man with carcinoma of the stomach, the author saw a nodule in the rectus abdominis which was thought by others to be a gumma. More rarely, they appear by the hematogenous route, as illustrated by the example mentioned on p. 710 of an ossifying metastatic carcinoma nodule in the musculature of the thigh, coming from a primary gastric carcinoma. The tumor masses may form diffuse infiltrates or disseminated nodules. The cells spread in the internal perimysium, but also enter the sarcolemma tubes, and thus compromise the muscle substance or destroy it in spots (Ribbert). Tumors which come from the periosteum or the bones such as osteomas, sarcomas, chondromas, (p. 1206), frequently spread into the neighboring muscles by continuity, or by discontinuity as though seeded.

The reactions of muscles to the presence of malignant tumors are degenerations, regenerations and inflammations. Rows of giant cells appear from the primitive bundles and from connective tissues (Fujinami, see Busse).

Animal Parasites

Trichina (trichinella) spiralis (Owen) is the most important parasite of muscles. It was first seen by Paget and Owen in 1835, and recognized

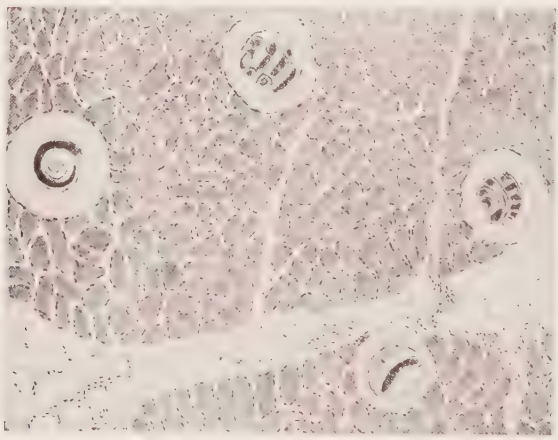


Fig. 996.—Cross section of muscle with numerous encapsulated trichinae cut at various levels. Pectoral muscle of man. (High power.)

as the parasitic cause of a serious disease by Zenker in 1860; by microscopic investigation of the muscles of an individual diagnosed clinically as having typhoid fever, he found "fresh migration of trichinae."

Trichinae reach the gastrointestinal tract of a host *e.g.*, a human, as developed worms (round worms) in raw or insufficiently smoked or salted pork. After their surrounding capsule is dissolved by the gastric juice, they reach sexual maturity in about $2\frac{1}{2}$ days; these intestinal trichinae

are white, stringy, just visible worms. Invasion into the intestinal tract is followed by the symptoms of gastrointestinal catarrh of varying severity according to the degree of infestation. If massive, vomiting and profuse diarrhea occur, and pea-soup-like stools are passed as in typhoid fever, or rice-water stools as in cholera. The sexually mature intestinal trichinae perform coitus in the small intestine; the males are one to two millimeters long and die soon after; the females, about three millimeters long, give birth to embryos about seven days later, each female producing from one thousand to thirteen hundred embryos. The pregnant female trichinae then penetrate the intestinal wall, especially the upper small intestine as Cerfontaine showed in rats and Askanazy in rabbits, to the submucosa, where they lie free in the tissues or in lymph vessels; from here, thousands of motile embryos, from 0.09 to 0.1 mm. in length, enter the lymph stream directly. Isolated individuals may be found in various other situations in the intestinal wall and free in the peritoneal cavity.

Trichinosed animals may infest others through their feces (Hoyberg, Rabiger), but this is disputed by Staubli; intestinal trichinae probably cannot be infective, at least only insofar as the embryos may be transferred into two or more hosts.

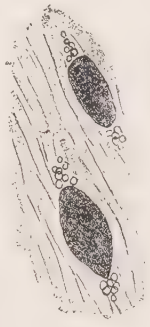


FIG. 997.

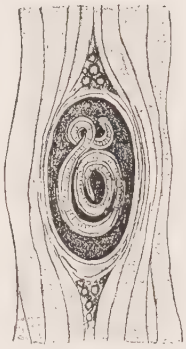


FIG. 998.

FIG. 997.—Calcified trichinae in human muscle; fat infiltration at poles. (× about 25.)

FIG. 998.—Cross cut through an encapsulated trichina in muscle after dissolving the calcium salts. (High power.)

From the intestinal wall, in which they produce no particular changes, the embryos reach the thoracic duct through the lymph stream and thus enter the blood; they are carried passively into various, but only cross striated, muscles. They have been found in the capillary blood of the heart muscle, but have never settled in this structure (Hertwig, Staubli, de Jong and others). They soon leave the capillaries and actively enter the interstitial tissue, and then through the sarcolemma tubes to their goal, the primitive fibers (stage of invasion). This takes about two weeks (nine to ten days) from the time of infestation. Most of the animals are collected at the ends of the muscles where they are stopped by connective tissue.

The duration of life of the intestinal trichina is from 5 to 8 weeks; propagation takes place for this length of time with resulting successive invasions of the muscles.

The complete development of the muscle trichina takes place in somewhat over 14 days when they are from .7 to 1 mm. long and have a pointed head and a blunt tail. Thus three weeks after infestation, the trichinae

which are now infectious to other hosts, lie in the swollen sarcolemma tubes, the contractile content of which they destroy, as they gradually roll up spirally toward their posterior side. Between the convolutions of the worm, is a finely granular mass representing the albuminous and fatty destruction products of the muscle substance (fig. 998). The muscle nuclei undergo proliferation. In the beginning, the trichina is surrounded only by the sarcolemma. Even in this stage, there develops a focal interstitial myositis with many eosinophilic cells. Later it becomes surrounded by a chitin-like, bright, laminated, striped, spindle or lemon shaped capsule which it supplies itself, and which, in addition to surrounding the parasite, surrounds portions of the muscle substance and nuclei; this is then surrounded by a fibrous covering supplied by the growing bordering connective tissue; the sarcolemma disappears, the muscle fibers undergo atrophy. The interstitial inflammatory foci are gradually absorbed. One capsule may surround one or a number (2–5) of trichinae. At the end of about a year, fat cells in the connective tissue develop at the poles of the capsule. At first the capsule is entirely transparent and in this stage can be recognized only microscopically, *e.g.*, in the fresh lethal cases of trichinosis in humans; after a longer or shorter time (3 to 9 months), calcium is deposited, beginning at the poles, and thus the structure is rendered macroscopically visible. Calcified capsules appear grossly as short, thick, yellow white points and stripes, clearly visible in many muscles, *e.g.*, those inserting on the exterior of the larynx; microscopically (enlarged 25 to 50 times), they appear cloudy and dark gray to black by transmitted light (fig. 997), and bright white by direct light.

In pigs, the capsule is practically never calcified and is difficult to see with the naked eye, since pigs are usually slaughtered under a year old, and, time has not been allowed for the deposition of the calcium salts. Pigs practically never die of trichinosis, and seldom show any symptoms. See pp. 2038 and 2040 for hyalin degeneration in trichinosed muscle, and for myositic round cell infiltration.

In examining for trichinae, small bits of the red part of the diaphragm, the intercostal muscles, the abdominal muscles, the laryngeal muscles are cut in the direction of their fibers; the bits are teased apart, flooded with potassium hydroxide solution which swells and destroys the muscles while the trichina are retained; they are then examined in calcium solution or glycerin with a magnification of about fifty. To examine thicker pieces of muscle (1 cubic centimeter) with low powers, they should be compressed between strong pieces of glass.

Muscle trichinae remain viable in their encysted stage surrounded by their calcified capsules for long lengths of time, even years (31), and are capable of development, although isolated individuals die even before they

are encapsulated (Ehrhardt). This may be called the resting stage with latent clinical symptoms. In feeding experiments with rabbits, intestinal trichinae, embryos, and then muscle trichinae are developed in seven days. Rats are very susceptible, die quite readily of intestinal trichinosis, although the latter are usually not infectious; trichinosed rats may infect pigs. Furthermore, guinea pigs and cats are also quite susceptible. Dogs are resistant, but not entirely immune. Even after the death of the host, the trichinae do not die immediately but remain living for weeks, even in decomposed meat. Temperatures of sixty-five to sixty-nine degrees centigrade kill the parasite. If an encapsulated trichina dies, it may be dissolved alone or with its capsule, or may become calcified; spontaneously dead trichinae are folded and dried. Afterwards the growing, surrounding connective tissue in the capsule infiltrates both the capsule and the parasite and eliminates the latter (stage of elimination); the final residue consists of fibrous nodules and strands (Langerhans, Ossipow). More marked invasion is followed by high degrees of atrophy of the muscles and the formation of cicatrices, particularly where the muscle becomes a tendon. Later there may occur complete regeneration (Kuttner-Landois).

Invasion of the trichinae is more vigorous in humans and animals in situations particularly active and well supplied with blood, such as the diaphragm, the glottis (particularly the dilaters, Hart), the eye muscles, muscles of the tongue, intercostals, abdominal, back, especially near the bones and insertions of the tendons; it is usually milder in the muscles of the extremities. The reason that the trichinae do not settle in the heart muscle is said to be because of the extraordinary tenderness of the sarcolemma of heart muscle fibers (Hertwig). Virchow found trichinae in the mesenteric lymph nodes, Fiedler, Staubli in the blood, Heller and Staubli in animals in the thoracic duct fourteen days after feeding.

Trichinosis is becoming more and more uncommon since the introduction of careful inspection of meats; it begins in typical cases with gastrointestinal catarrh and abdominal pains. In the second week or later, there appear the muscle symptoms, consisting of swelling, severe pain, paralysis, trophic disturbances, and almost always absence of knee jerks (Eisenlohr). The picture may simulate that of neuritis and polymyositis. There is pain upon moving the eyes, even strabismus, and difficulty in chewing and swallowing. Involvement of the muscles of respiration may lead to fatal aspiration pneumonia. Edema, the third chief symptom, occurs almost without exception, and very early (from the seventh day onward), in the eyelids and in the face, particularly under the eyes, but later in other situations such as the skin of the upper and lower extremities. Fever and prostration are practically always present to considerable

degrees; the intermittent fever is similar to that of typhoid fever, (the famous first case of Zenker was mistaken for typhoid), but it is of longer duration. The entire duration of the disease is four to six weeks. The mortality varies, but occasionally is quite high. Death occurs usually in four to six weeks; at autopsy, as a rule, are found fatty changes in the parenchymatous organs, particularly the liver, but also in the kidneys and irritation of the serosae, which is believed to be an expression of the

FIG. 999.—Miescher's tubule (left); encapsulated trichina with surrounding eosinophilic ecll infiltration (right). Muscle from pig. (Medium power.)

FIG. 1000.—Numerous cysticercus cellulosae in muscle of upper arm (biceps); ½ nat. size.

activity of the toxins of the trichinae (Romanowitsch) and may influence the clinical course (Gruber). In children, trichinosis usually runs a milder course.

For pseudotrichinosis, see p. 2040.

The blood eosinophilia appears about eight days after infestation, or somewhat later, and is important diagnostically; the eosinophiles come from the bone marrow (see Staubli, lit., Knorr, Bittner, and p. 197). The local foci attract many of these cells, perhaps by chemotaxis, or to attempt removal of the toxins of the trichinae (Huebner, animal experiments). Staubli also points out that diametrically opposed to (uncomplicated) typhoid fever, trichinosis is accompanied by a leucocytosis, caused

not alone by eosinophilic cells, but also by neutrophilic leucocytes (for more clinical details, see Strauss).

In the diagnosis of the meat of pigs, differentiation must be made from the common sarcosporidia in the form of so-called sarcocystis Miescheriana, which were first found in the ordinary wild mouse. These are macroscopic yellow white, microscopic dark and granular, oval, rounded or tubular-like, structures about 1 millimeter in length, found particularly in the muscle fibers which still show cross striae (v. Hessling). Some of them lie in the connective tissue and were formerly looked upon as being parasites of the muscle fibers (Braun). The tubes (fig. 999), consist of a membrane surrounding innumerable bean-shaped bodies (sporozoites); oblique septa divide the structure into numerous cavities or chambers. They may be mistaken by the inexperienced for calcified encapsulated trichinae, which, however, may also be present (fig. 999), or for dead or calcified cysticerci, particularly because the tubes are calcified.

Cysticercus cellulosae is relatively frequent in the muscles (lit. Danielsen and Kuttner-Landois). The pectoralis major and the biceps are preferred. They produce vesicles in the intermuscular connective tissue, which become spindle-shaped from the pressure of the neighboring muscle bundles (fig. 1000). They bring about a mild interstitial myositis. Usually the cysticercus is isolated, and present as individuals, but in more massive infestation, they are numerous, and the nodules may be felt in the muscles through the skin; usually, however, these cases have deposits in the subcutaneous tissue. As a rule such cases run a latent course. See p. 848 for the natural history of the cysticercus and its relationship to taenia armata.

Echinococcus of the muscles is very rare; usually they are solitary, and very rarely, multiple.

Sites of predilection are the posterior parts of the trunk, the inner side of the thigh, the neck, the upper arm. Muscle echinococci are said to be the more frequent the nearer the abdomen the involved muscle is situated. It is even believed that echinococci may wander through the intestinal wall into the retroperitoneal tissue, and thus reach the periphery through the sheaths of the vessels. In other cases it is believed there is a transport of embryos through the blood vessels into the muscles.

(Lit. by Kuttner-Landois, and by Thorel and Baradulin.)

CHAPTER XII

TENDON SHEATHS AND BURSAE

Diseases of the Tendon Sheaths and the Tendons

The tendons are surrounded by the tendon sheaths, in which they glide to and fro, lubricated by the synovial fluid. Diseases of the sheaths and the bursae in general are quite similar to those of the synovial membranes (see under joints).

Inflammations, tenosynovitis, are observed after injuries, crushes, overexertion, or as extensions by continuity, inasmuch as inflammations of the hand or foot spread through the tendon sheaths. More rarely they appear by hematogenous routes as in the so-called rheumatic inflammation. They may come from inflammatory foci in other situations, or spontaneously.

In acute tenosynovitis, there is a serofibrinous or a purely fibrinous exudate (*a*) in the tendon sheaths. The purely fibrinous forms are also called dry, acute tenosynovitis.

If the hand is laid on a diseased tendon, a sensation of grating is felt when the tendon is moved, due to the rawness of the surfaces (crepitating tenosynovitis). It is observed most frequently following overexertion of the forearm, (for the foot, see Weiss; for other ideas, see Troell who believes that the most important process is extra-tendonous). (*b*) Suppurative tenosynovitis usually arises by continuity from the neighborhood, *e.g.*, from a panaritium. It may, however, appear from hematogenous sources. Both forms, the fibrinous and the suppurative, occur, *e.g.*, in gonorrhea, in which the suppurative inflammation is apt to cause serious damage to the tissues. Jacobi and Goldmann were able to demonstrate gonococci in the tissues. Gonorrheal tenosynovitis is occasionally isolated, but often appears in conjunction with inflammation of the joint, occasionally also with multiple metastases. In the foot and hand, it usually is simultaneous with inflammations of the joints, (p. 1238); D. Nasse is of the opinion that the stubborness of this combination and the persistence of disturbances of movement are characteristic in contrast to rheumatism of the joints. See also gonorrhea of the bursae, p. 2065.

In severe tenosynovitis, the ordinary pyogenic cocci are found; the tendon may undergo necrosis; it becomes cloudy, swollen, thready, dis-

colored and shrunken, occasionally dry; even without this, it may adhere to its sheath.

Chronic tenosynovitis from long continued irritative factors may be initiated by marked serous exudation. This hydrops or hygroma of the sheath is seen most frequently in the hand, particularly the flexors, on the back of the hand and the flexor surfaces of the fingers. In the palm of the hand, the dilated sheaths take on an hour glass shape because the ligamentum carpi volare is stretched over them. The so-called rice bodies often appear in these hygromas; they are also called corpora oryzoidea, when the disease is, as a rule, of a tuberculous nature (see below, under tuberculosis).

Of the chronic non-tuberculous varieties, there may be recognized two forms: (*a*) the rheumatic, characterized in typical cases by lymphocytic infiltrates arranged in follicular manner with germinal centers (A. Vischer), and (*b*) the stenosing, first described by de Quervain along the styloid process of the radius, characterized by an increase in the connective tissue undergoing hyalin change, and by a rich vascular supply. This is differentiated from ordinary tenosynovitis by the fact that the round cell infiltration disappears completely. For more details, see Keppler and A. Vischer, (lit.).

In gout (p. 1252), urates are deposited, often to considerable extent, in the tissues of the tendons and sheaths, and chronic inflammations progress with exudative necrosis of the tissues, and reactive new growth. The tendons and sheaths may be infiltrated by a cellular tissue, sufficient to fill the sheaths.

This granulation tissue, perhaps containing giant cells, surrounds the urate deposits and even infiltrates into them at times (uric acid proliferating tenosynovitis, see p. 1253).

Tuberculosis of the tendon sheaths appears most frequently by continuity from a tuberculous joint or bone. More rarely, it is primary, particularly in the forearm (sheaths of the flexors of the hand), and on the posterior aspect of the hand. Tuberculous granulations are established in the sheaths, and are accompanied by serous (*a*) or sero-fibrinous (*b*) exudate. The granulations may even become (*c*) tumor-like, with minor degrees of exudation, and may produce a fungus form of connective tissue which later shrinks. In other cases (*d*), the caseous granulations produce pus, *i.e.*, a cold abscess, perhaps with fistulae communicating with the exterior. The form (*a*) accompanied by purely serous exudation is also called serous hydrops or tuberculous hygroma, whereas the sero-fibrinous variety (*b*) often produces a rice body hygroma with large numbers of these rice bodies or melon seed-like free bodies causing gratings and rubbings in vivo; details were sufficiently discussed under the analogous affections of the joints (p. 1257).

See Forgue and Etienne (lit.), and illustrations of tendonous synovitis with rice bodies.

Syphilis.—In tertiary syphilis, gummatous nodules in the tendon sheaths have been described, varying from the size of cherries to walnuts and movable with the sheaths, or in the tendons themselves and spread to the sheaths (rare), (lit. by Ullmann).

True **tumors** of the tendon sheaths are very rare (Bellamy, lit.). The author examined a sarcoma which appeared like a tendon sheath hygroma, and was composed of short, thick spindle cells, some of which were arranged in rows; this tumor was the recurrence of a tendon sheath hygroma removed two years previously from the region of the first metacarpal on the dorsal side of the right hand of a forty year old woman. Literature concerning sarcoma by A. Rosenthal.

The tendon sheath tumors known as "myelomas" (Heurtaux, Bonjour, Malherbe and others), are known to the author through three observations; in two cases they were from the external malleolus of the foot, in the third from the flexor surface of the small finger (Hartert). Clinically, they are relatively benign, liable to recur locally, but produce no metastases. They are lobulated, and on the fingers may reach the size of a hazel nut, in other situations, of a hen's egg. Histologically, they have a sarcomatous appearance; spindle cells are found with enormous (myeloplax-like) giant cells, yellow brown pigment giving the iron reaction, hyalinized vessels and interstitial substance and whole nests of cells quite similar to the foamy "xanthoma cells" (see under skin), in which doubly refractive substances can be demonstrated, and which may also give a macroscopic cloudy white appearance to the remaining yellow brown section surface. In general, they are quite similar to the tuberous xanthomas (see under skin). Indeed, there are cases of xanthomatosis, in which multiple tumors of the skin are found with similar tumors in the tendon sheaths and the capsules of the joints (thus, for example, case of Arning); the fascias and the periosteum are also points of origin of these tumors (Landois and Mont Reid). The designation "myeloma" because of the giant cells (myeloplaxes), is not to be recommended. They are also spoken of as pigment-containing giant cell sarcomas, or fibrohemosiderin sarcomas, gigantocellulare xanthomatodes, etc., (A. Rosenthal, Hartert, Spiess, lit.). It has also been thought possible that, when these tumors appear after one or more severe traumata, leaving out of consideration other changes in the tissue, cholesterin precipitates are produced (increase of the blood cholesterin is probably necessary), which act as irritants on the surrounding tissues; they would therefore be granulation tumors (Weil, Arzt). After striking the Achilles tendon of rabbits, Hoessli, in animals with hypercholesterinemia, found

xanthelasma-like structures of young fibroblasts containing cholesterin. Literature by A. Rosenthal, Spiess, Alary, Tourneux, Fleissig, Arzt, and under skin.

Diseases of the Bursae

The bursae are spaces filled with synovial fluid, and surrounded by a flat connective tissue membrane. They originate as spaces in the interstitial tissue (Disse). There are differentiated: subcutaneous (Langemak) and deep lying bursae; the latter are in the neighborhood of joints, but do not communicate with them. They are placed like pillows where muscles or fascia pass over bones, or where there is much movement between muscles, fascia and skin. Ordinarily they are found only at typical situations, but since they are structures developing from use, they may appear in atypical places under such special circumstances as chronic traumatic, or inflammatory irritations (B. Martin).

Acute inflammations of the bursae with serous, sero-fibrinous, or suppurative exudation are usually traumatic in origin. They may, however, be hematogenous, as in gonorrhea, when a very stubborn pain, the so-called achillodynia (Albert), is produced from a bursitis of the tendon of Achilles (Nasse, Nobl). According to some, the pain is caused by an inflammation of the Achilles' tendon itself (Jacoby), or by a periostitis of the os calcaneum. Kaumheimer (lit.) saw a purulent pneumococcus bursitis in pneumonia.

A chronic bursitis, a hygroma, or a hydrops follows an enlargement and accumulation of fluid in a normal or newly formed bursa. This is seen particularly often in the prepatellar bursa of males who work hard, and females as the housemaids' knee; an irregularly outlined cavity filled with fluid is found in the connective tissue; at first it is always traversed by fibrous bands and trabeculae, and is produced by cystic change in the prepatellar bursa.

Other localizations of chronic bursitis are: subacromial, the bursitis which produces the pain called rheumatism of the shoulder, neuritis or periarthritis (Codman, lit.); bursitis of the olecranon with a hygroma is often seen in people in mountainous countries.

The wall of the hygroma is irregular in the beginning, and tender, but later it is thickened and hyalin, and, in typical cases, the inner surface is nodular and covered with dendritic, hyalinized, fibrinoid, pear- or egg-shaped tags and nodules, plica and trabeculae, which produce meshes, and pass through the cavity or merely enter it. Pedunculated portions of fat tissue or pieces of cartilage, occasionally become free and produce foreign bodies analogous to joint mice (p. 1274). Partial calcification also occurs. Such cases (fig. 1001), were formerly called bursitis proliferans (Virchow),

but in the majority of cases, as pointed out by Graser, it is not an inflammatory productive process, but rather one of degeneration. The cavity is stretched after degeneration and solution of parts of the wall; the prominences and trabeculae are parts which are better nourished and resist destruction. Langemak, who is disputed, however, by Kreuter, identifies this process with that of the formation of ganglia (p. 2067), and believes both are the products of secondary solution of fibrous hyperplastic connective tissue (Ricker). The hyperplasia is said to occur from arterial hyperemia.

But Langemak remarks correctly that the designation "hygroma destruens" (Graser), is not to be recommended. From the examination of many

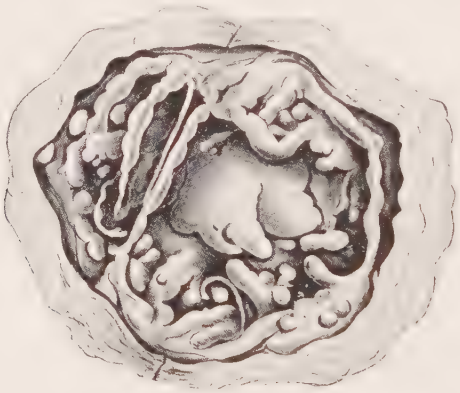


FIG. 1001.—Prepatellar hygroma, man 45 years old, laborer; opened and exposed; $1\frac{1}{12}$ nat. size.

cases at various stages, especially early, the author has reached the conclusion that the analogy to the process in ganglia (and tendon sheaths) is well taken.

The content of a hygroma is at first thick, slimy, honey-like and contains much albumen; later it is thin, fluid, and serous. Secondary hematomas may appear (bursa hemorrhagica). If corpora oryzoidea (so-called crepitating ganglia), are found, it is usually the symptom of a tuberculous disease of relatively benign prognosis (hygroma tuberculosum, see p. 2063). The exudate is usually clear and serous. But the tuberculous hygroma may appear in the form of a luxuriant, fungoid, markedly caseating, granulation tissue mass. Reinhardt differentiates from the more frequent form with tuberculous granulation tissue, a primarily sclerosing tuberculosis of the tendon sheaths in which little specific products are produced, but rather a tough connective tissue. Moreover, infection may occur secondary to disease of the neighborhood (bones, joints), as well as primarily, which vice versa may spread to the neighborhood (Rubesch); see also Stahr.

The so-called **ganglion** which usually appears in or near the wrist joints (carpal ganglion) or on the back and middle of the hand, more rarely in the middle of the foot and in the knee joints (lit. by Jastram), is a cyst-like structure produced by mucoid degeneration of fibrous tissue.

Formerly it was believed that a ganglion was a hydrops of the tendon sheaths with thickening of the skin, or a diverticulum-like projection of the tendon sheath, or bursa, and comparisons were drawn with the hernias of the synovial sacs of the joints. According to Ledderhose, Ritschl and Thorn, however, ganglia originate from a colloid-like degeneration in the hyperplastic connective tissue around the joints, which is produced by traumatic causes (pressure). The connective tissue, increased by use, is softened, usually from rubbing over bone. Small colloid foci are formed with mucoid cavities in the connective tissue, which gradually undergo confluence to a cyst which may reach the size of a pigeon's egg, and be filled with thick, slimy, glassy colloid. Absorption of the cyst contents may lead to healing of the ganglia.

On the other hand, Payr believes that the carpal ganglia are traumatic, inflammatory, softening cysts in the para-articular connective tissue, not as the result of pressure but of the action of pull. In almost all cases, he found giant cells in their content and also in their wall (Franz). Floderus (comprehensive lit.), believes that ganglia come from the tissues around the joint, which have remained unused during the ontogenesis of the joint, and which have lost more or less of their early structure; thus he calls them joint tissue tumors or synovial cysts (arthromas). Their further development he explains as does Ledderhose, *i.e.*, from softening.

Tumors of the Tendon Sheaths are Rare.—There occur sarcomas, of the round or spindle cell, occasionally also giant cell (Fritsch), varieties; there have also been described myxomas, endotheliomas, and papillomas. Point of election: prepatellar bursa. (Lit. by Adrian and Martina, Bolognesi, subdeltoid bursa, lit.)

CHAPTER XIII

SKIN

(For the embryology, see Pinkus in Handb. der Entwicklungsgeschichte von Keibel-Mall 1910, first volume.)

Post Mortem Changes

The most important are:

1. **Livor,** or hypostatic discoloration, which begins one to four hours after death and reaches fullest development in twelve to twenty-four hours; it occurs in practically every body, even in those who died with severe anemia. It appears in the dependent parts, *i.e.*, when the cadaver lies on its back, in the back and the posterior surfaces of the extremities and the neck, as a bright red to blue red discoloration, in spots, fairly sharply bordered with their edges flecky and marbled; the blood sinks to these parts. This hypostasis also appears in the internal organs. Livor is particularly well marked when the blood remains fluid, as in all forms of death by asphyxia; the spots are then blue red, but in carbon monoxide and prussic acid poisoning they are bright red. The so-called "wandering of the livor" can be observed when the position of the cadaver with fresh livor is changed, whereupon the livor follows the dependent parts. An older livor from which imbibition with dissolved blood coloring matter has taken place, does not show this change (Bartel). Parts which have had pressure exerted upon them remain free; they appear as bright specks and stripes on a blue red background. Differentiation of hypostasis and hemorrhage on section: in hypostasis the blood can be washed away with water, after which the section or incision is pale. In hemorrhages, on the other hand, the tissues are infiltrated by blood and the blood does not flow away on playing water upon the parts.

2. As a result of decomposition, there appear (*a*) diffuse spots discolored red brown to green, and dendritic stripes, particularly along the veins of the skin; they gradually become broader. They result from diffusion of the blood coloring matter following hemolysis. The edges of the stripes are obscure; vesicles of gas may appear in them. In addition to the skin, the subcutaneous tissue may also take part in these changes; gas bubbles appear in this tissue. (*b*) The discoloration of the abdomen, also called pseudomelanosis, is of a green to a green blue color,

first appearing in the right hypochondrium where the cecum lies close to the surface. The intercostal spaces and the supra- and infraclavicular fossae may also show this discoloration. The gases of decomposition and the gases from the intestines diffuse (hydrogen sulphide and ammonium sulphide) and are combined with the coloring matter of the destroyed blood corpuscles into sulphur methemoglobin (Hoppe-Seyler, Harnack). (*c*) Blebs appear in the skin as decomposition progresses. These vesicles may burst, and the situations become dry and parchment-like. (*d*) In the so-called post mortem emphysema, the distended skin crepitates. The most important cause is the formation of gases of decomposition in the subcutaneous tissues.

3. **Appearance of Desiccation.**—Wherever smaller or larger pieces of epidermis have been removed either before or after death, the situations dry rapidly, take on a brown color, and become leathery, tough, transparent, and somewhat sunken.

Circulatory Disturbances of the Skin

Hyperemia appears diffusely or as a circumscribed redness which disappears on pressure with the fingers; it varies in color from pale rose red to dark blue red, as a symptom of congestive hyperemia, which is the reverse of that in the living. Simple hyperemia of the skin disappears after death. Clinically, small hyperemic spots are called roseolae; more extensive areas are designated as erythema. These represent the mildest grades of inflammations of the skin and will be discussed under that heading. Venous hyperemia which may involve large areas of the skin is called cyanosis, and can be recognized in the cadaver. The skin becomes red to dark blue. A small cyanotic spot is called livedo.

Erythrodermia congenita may be universal or partial (head, hands); the skin takes on a brown red or copper red color (Indian skin) and may desquamate or not. Microscopically there are increase and dilatation of the capillaries, pigments in the epidermis and corium, and thinning of the epidermis (lit. by Rasch, Schonnefeld).

(*b*) Anemia of the skin, as in those dying of hemorrhage, or in those individuals with chlorosis, can be observed even post mortem by the pale, waxy, often somewhat greenish skin discoloration.

(*c*) Edema of the skin (anasarca) consists of saturation by watery fluid, particularly of the subcutaneous tissues and less in the tough actual skin. The swollen skin, cold, pale in simple edema, and red and hot in inflammatory edema, is smooth on the surface; the pressure of a finger leaves a dent.

Simple non-inflammatory edema may appear as a stasis following local mechanical interference with the blood stream (as *e.g.*, Bier's hyperemia).

or as a symptom of a general edema in heart disease, or as a symptom of kidney disease. In the latter, the localization and intensity often vary; it is thought to be less a direct disturbance of the function of the kidneys in excreting water and salts than an indirect damage to the vascular apparatus and the tissues of the subcutis and serous cavities, which leads to the collection of large masses of sodium chloride-containing fluid. The edema disappears by entrance into the lymph vessels and resorption into the blood; this may be followed by polyuria. Direct toxic edema of the skin occurs in insect bites and to greater degrees in snake bites; the boundary between simple and inflammatory edema disappears in these cases. Collateral simple edema is seen in the neighborhood of inflammatory foci. Angioneurotic edema results from a changed innervation of the vessels (irritation of the dilators, paralysis of the constrictors). In this category belongs the circumscribed edema of the skin (Quincke), which, because of its remarkable suddenness, is called acute paroxysmal edema. It is a familial disease and may lead to death from edema of the glottis (Harbitz, lit.). In complete obstruction of the lymph nodes from carcinoma or suppuration, edema occurs in the arm, the leg, scrotum, labia, etc. Chronically edematous tissues are more susceptible to infection; thus, edema of the scrotum is frequently followed by phlegmons, and erysipelas occurs in edematous legs.

The production of an infiltration anesthesia depends upon an artificial edema. See below, concerning infectious malignant edema.

In **myxedema** (p. 526), the skin, particularly that of the face, is pale, dry, elastic and swollen. This swelling does not disappear upon pressure with a finger. It is not caused by a collection of serous fluid, but is an infiltration of half fluid, slimy masses into the connective tissue meshes of the skin. The glands of the skin then cease to secrete.

Malignant edema runs a very unfavorable course, is caused by the motile bacillus of malignant edema (R. Koch, Gaffky), an anerobic pathogenic organism of decomposition, which infects the soil, among other places. The subcutaneous intermuscular connective tissue is saturated with a purely serous, yellow, or lemon yellow, or sero-sanguinous fluid in large amounts. This fluid contains the bacilli in masses; there are few or no gas vesicles, and it is without odor. No pus is formed. When the organisms are of high virulence, they may produce gas, so that such cases are quite similar to gas infections (p. 2042); but the destruction of tissue which characterizes the latter disease is lacking. The bacillus of malignant edema is quite different from the chief cause of gas infections, namely, Eug. Fraenkel's gas bacillus (see p. 2042; consult also Ghon, Eug. Fraenkel, v. Baumgarten). See also v. Werdt concerning malignant edema.

The so-called nutritional edema is based on qualitatively and quantitatively insufficient nutrition (general atrophy), with a tendency to edema. Pollag (lit.), Schittenhelm and Schlecht, explain the edema by assuming there is damage to capillaries, but this point is still debatable (Prym, lit.). Paltauf believes cases of chronic inanition (marasmus ex inanitione), which do not show edema, but which, as many cases of true edema, end quickly in death from central vasomotor paralysis, are fundamentally the same as nutritional edema. Hydrops universalis congenitus with erythroblastosis has been described on p. 240. This edema is universal, in the form of an anasarca and hydrops of all the body cavities. Etiologically, this form is obscure; Schridde believes it is due to anemia, Chiari and others to maternal toxins, others to toxins from nephritis in the mother (Nyhoff, Liegner, lit.); according to Ludwig, the fetus produces edema in the mother from its metabolic processes, whereby the fetus itself becomes edematous secondarily. Lahm found syphilis in one case. In other cases disturbances of the blood and lymphatic circulation of the fetus are the causative factors; thus, endocarditis, a small foramen ovale, and obliterated ductus Botalli (Alexandrowsky, lit.), thrombosis of the umbilical veins, thrombosis of the inferior cava (Link, lit.). Furthermore, edema and hyperplasia of the placenta, as well as hydramnion (p. 1720), with edema of the membranes, have been considered responsible for hydrops universalis (lit. by Alexandrowsky).

Hemorrhages in the Skin.—Hemorrhages into the skin and the subcutaneous tissue appear in the form of small circumscribed punctate or larger spots (petechiae), which do not disappear on pressure with the finger, or as stripes (vibices), or nodules (lichen hemorrhagicus or purpura papulosa), or as irregular spots from the size of a silver dollar to a dinner plate (ecchymoses), or as larger ecchymomas or hematomas, situated in the corium and subcutaneous cellular tissue, but sometimes in the epidermis also. Spontaneous hemorrhages are grouped under the general name of purpura.

The site of superficial hemorrhage is chiefly in the corium, particularly the stratum subpapillare, from which the blood escapes into the layers of the epidermis. If the hemorrhage is of any great extent, the epidermis may be pushed upward in the form of vesicles. If the blood enters the sweat glands from the surrounding capillaries, it may ooze from their ducts (hematidrosis); this is a rare form of neurotic hemorrhage (lit. by Marchand). The fate of smaller hemorrhages, as a rule, is rapid absorption, following chemical changes in the blood coloring matter. The red spots become blue red, green, yellow to brown. The pigment, as a rule, is carried away in the lymph channels and the regional lymph nodes become pigmented. The blood between the epithelial cells is gradually

pushed away along with the physiological desquamation of the epithelium; where the pigment remains, there is a temporary black spot.

Etiology.—Hemorrhages into the skin occur: (1) from traumata such as crushing, contusions. (2) As the so-called symptomatic hemorrhages during the course of many acute infectious diseases such as variola, scarlet fever, endocarditis, septicemia, rheumatism, spotted fever (for the histological peculiarities of the latter, see Eug. Fraenkel, and p. 122), typhoid fever, pneumonia, various kinds of meningitis, bubonic plague and others (symptomatic purpura, see Lenhartz); they appear as spotted, punctate, or stippled, or nodular, or vesicular hemorrhages in the skin; most of them are surely metastatic in origin (Eug. Fraenkel), and many are accompanied by necrosis of the connective tissue and inflammatory changes, particularly in meningococcus meningitis (Eug. Fraenkel, Gruber, Pick, lit.).

> **Translator's Remarks.**—But extracts of organisms alone, *e.g.*, pneumococcus, may produce purpura. Pneumococcus extracts are thrombolytic and hemolytic; when the number of platelets diminished to less than 500,000 per c.mm., purpura usually developed in white mice; see Julianelle, L. A., and Reimann, H. A., J. Exp. Med. 63: 87: 1926.

(3) Toxic purpuras occur in intoxications, as from iodine, bromine, antipyrine, phosphorous, in jaundice, snake bites and others; (4) cachectic purpuras occur in serious disturbances of nutrition such as anemia, leukemia, carcinoma, multiple sarcomas, Bright's disease and uremia; in the latter, they are probably due to a damage to the resistance of the vessel walls. Senile purpura appears especially in the lower extremities following circulatory disturbances of marked arteriosclerosis. In a certain number of cases of type (2), the hemorrhages occur from obstruction of smaller skin vessels with bacteria, or in the above named autointoxications and the ordinary poisonings, from damage to the vessel walls, from the toxins produced by the bacteria in the neighborhood (Leube). (5) Hemorrhages are symptoms of diseases of obscure origin called essential or idiopathic purpuras. Many of these cases are infections (hemorrhagic infections), as demonstrated by finding known bacteria (streptococcus pyogenes) or newer species, particularly bacilli (Petrone, Tizzoni and Giovanni, W. Koch and others), in the extravasations and in the blood; as further support of this statement, it is known that certain forms of purpura hemorrhagica allow of vaccination into other individuals. (7) Neuropathic purpura occurs in hysterical individuals (Binder); thus also are explained the "wonderful" hemorrhages in the stigmatised. Literature by Marchand.

Various forms of essential purpura may be differentiated. In general, they exhibit a tendency to spontaneous hemorrhages in the tissues and into the surface of the skin and mucous membranes. In the mildest cases of purpura simplex the spots of blood are confined to the skin, chiefly of the extensor surfaces; the bites of fleas (purpura pulicosa), may be mistaken for a purpura simplex; usually they are accompanied by pain and swelling of the joints (purpura s. peliosis rheumatica).

Purpura hemorrhagica (morbus maculosus Werlhofii), usually begins with a high fever and runs a subacute or chronic course; the hemorrhages into the skin are small, but innumerable, and, according to the investigations of Niekaus who used a microscope for studying skin capillaries, are produced from thrombotic-embolic processes in the upper subpapillary capillaries; hemorrhages also take place into the mucous membranes and the internal organs such as the kidneys, the brain, the omentum, as well as in the free surfaces of the nose, the kidneys, the intestines (lit. by Immermann). According to Steiger, the coagulating ability of the blood is markedly diminished, the viscosity is reduced, and the count of blood platelets remarkably small. Glanzmann, who looks upon the disease as an hereditary hemorrhagic thrombasthenia, which manifests itself externally as at hrombopenia (p. 186), found the coagulation time normal, but the coagulae were less firm and thus the arrest of hemorrhage disturbed; in hemophilia, on the other hand, the coagulation time of blood is increased, but the coagulum is firm and of normal consistence. Concerning thrombopenia in purpura hemorrhagica, see Fonio, Frank, Minkowski, Foerster, F. Sternberg. Acute leukemia may be confused clinically with this condition unless a blood examination is made (more details by Naegeli); the same is true of the rare cases of purpura variolosa (see H. Boehm).

Scorbutus belongs among these diseases; according to Kaposi, Leube and others, it is fundamentally no different from purpura; but coagulation time and the count of platelets are normal. According to F. Sternberg, damage or functional disturbance of the vessels is important in the origin of a purpura. Involvement of the gums (p. 550), is said to be pathognomonic, but this may not appear (Pfeiffer). These and the hemorrhages into the deeper tissues such as the muscles, the periosteum, the epiphyseal lines, etc. are characteristic of scorbutus. Aschoff-Koch believe there is a diminution of the cement substance of the supporting tissue inclusive of the vessel walls. Secondary hemorrhagic inflammation in the pleura, pericardium, endocardium, peritoneum and the kidneys is frequent. The etiology of scorbutus which is often seen as an epidemic in prisons, besieged towns, poorly provisioned ships, etc., has always been thought to be insufficient or improper nourishment, especially lack of fresh vegetables. It is now believed that scorbutus is one of the

avitaminoses (Funk) similar to beri-beri, pellagra, and perhaps also rachitis (see Bickel), diseases which appear when the food contains insufficient or improper vitamines—those substances extractable by alcohol, thermolabile, and contained in raw vegetables. See Bierich for literature concerning scorbutus. See also Moller-Barlow's disease, *i.e.*, scorbutus of children, p. 1166.

Inflammation of the Skin

General Remarks Concerning the Etiology of Cutaneous Inflammations.—The causes of inflammations of the skin are exceedingly varied: (*a*) direct external damage, from mechanical means, chemical, thermal, parasitic, etc., may be mentioned. They produce a primary essential dermatitis. (*b*) Indirect, internal traumata produce a secondary, or symptomatic dermatitis, *i.e.*, the inflammation arises from infectious organisms which have reached the skin from the blood to produce the so-called "pyamids," or better, infectious or toxic hematogenous dermatoses (Jadassohn; Eug. Fraenkel, lit.); or the toxins damage the vessels of the skin or the nerves, or the epidermis itself; or there are disturbances of nutrition and inflammation of the skin following the excretion of various poisons, as for example, iodine, antipyrine, salvarsan (Heyn), the poisons in pellagra, metabolic products as in uremia, etc.; or the dermatitis follows diseases of the central and the peripheral nervous system, irritations or disturbances of the nerves (compare the various forms of dermatitis in women with dysmenorrhea, lit. by Friedeberg). (*c*) Many inflammations of the skin are obscure in their etiology.

The general pathologic anatomic process in these various inflammations of the skin is exudative, but externally they manifest themselves quite differently in relation to their cause, form, course, and significance. The histological processes in the various forms of cutaneous inflammation will be discussed under the various disease processes. We will then discuss the elementary forms through which the efflorescences are manifested; but a short definition may perhaps clear the way.

These elementary forms are: spots (macula), a spotted change in the color of the skin; nodules (papula), solid cellular, or serous, half round, or conical protuberances of the skin up to the size of lentils; nodes (tuberculum s. nodus), a swelling resulting in nodules larger than papules; wheals (urtica), a solid efflorescence which is broader than it is high; vesicles or blisters (bulla), lie in the epidermis, and contain serous or sero-sanguinous contents; pustules are vesicles with purulent contents; impetigo, larger suppurative pustules. As further changes in form (secondary efflorescences), there occurs the formation of crusts and scabs, from the drying of pustules, excoriations, erosions, tears or rhagadae, and ulcers, which

vary in their depth and in their shapes and forms; furthermore there are desquamations, such as the flour-like desquamatio furfuracea, and the leafy desquamatio membranacea, which we will encounter later.

The Individual Forms of Cutaneous Inflammations.—[For dermatoses, exanthems, of monkeys and apes, see Fox, H., and Weidman, F. D., Arch. Dermat. and Syph. 7: 462: 1923 and Weidman, F. D., ibid 7: 289: 1923.—*Translator.*] The mildest inflammations of the skin are encountered in the exanthemata in measles, scarlet fever, in erythema exsudativum multiforme, and in urticaria.

(*a*) The exanthemata in measles and scarlet fever. These are mild forms of skin inflammations, characterized chiefly by their redness, and less by their exudative swelling.

In **measles,** which are characterized by the exanthem, fever and catarrh of the upper air passages, there appear rounded, flat or mildly raised, discrete, never generally confluent, bluish to yellow red spots with a central nodule, from the size of lentils to the joint of a finger, which become pale to yellow spots upon pressure (morbilli laeves). But there may also be formed fine red papules which are localized corresponding to the openings of the follicles (morbilli papulosi). The cause of measles is unknown, the portal of entry probably the nasopharynx.

It has been possible to transfer the disease to monkeys. Hlawa described protozoa-like structures between and upon red blood corpuscles. [For lit. and data regarding green aerobic diplococci, filter passers, work of Degkwitz, etc., see Cary, W. E. and Day, L. A., J.A.M.A. 89: 1206: 1927, with discussion.—*Translator.*]

See Ewing, Abramow, Mallory and Medlar concerning the finer histological changes in the skin.

According to v. Pirquet, the exanthem is an egotoxic reaction to the organism causing measles which remains localized in the skin capillaries; fever appears after an incubation period of ten days, and a catarrhal stage of prodromes lasting from three to five days, whereupon the exanthem makes itself noticeable first in the face, on the forehead and cheeks, from which it spreads to the neck, the shoulders, the back and the extremities. The time of greatest infectivity is in the prodromal or catarrhal stage, and during the first day of the exanthem, after which it is no longer contagious (Baur). The height of the exanthem is reached the first to the second day after its appearance (fifth to the sixth day of sickness); the temperature remains high for five to six days, and is followed by a critical fall. The height of the exanthem remains for twelve to twenty-four hours or even a shorter time. It then pales, leaving behind yellow to brown spots, and is followed by a furfuraceous desquamation beginning about the second week, occasionally accompanied by marked itching, which remains

for as long as fourteen days counting from the day of the eruption. Changes from this typical course are shown by morbilli vesiculosi, confluentes and hemorrhagici. This exanthem is quite disposed to run atypically when scarlet fever is implanted upon it (H. Risel). Very mild forms occur in sucklings, but forms which are quite important in the spread of an epidemic (Nassau). Koplik's spots, which, according to Mallory and Medlar, represent the early stages of changes in the epidermis, may become macerated, and secondarily infected, see p. 549.

Especially laryngitis and broncho-pneumonia may be mentioned among the complications of measles, since they frequently attack younger or weaker children. See Lutz, concerning the formation of thrombi, chiefly in the arteries of the lungs. See also p. 405.

In **scarlet fever,** the skin shows innumerable, fine, closely sown points or spots which are flat, or but slightly raised and seldom larger than the head of a pin or a lentil, appearing first in the neck and upper back, and then spreading over the trunk, the breasts, and the entire body. From confluence of the points, there practically always arises a diffuse punctate reddening, as if the skin had the dark uniform red color of scarlet fever poured over it; at the same time, it becomes swollen.

In differentiating measles: the face is less involved, but when it is, the mouth and the region of the chin usually remain free (Leube, and others). For the tongue of scarlet fever, see p. 548. The so-called scarlet fever line is an obliquely crossing linear furrow found upon the surface at the roots of the finger-nails which, as the nail grows, gradually reaches the free edge in about six months (Feer).

See Rach concerning the histology of the eruption in scarlet fever in which there are acute inflammatory foci with the production of serous or cellular or cellular hemorrhagic exudate; among the cells of the exudate, the polynuclear leucocytes are predominant. The scales consist of incompletely cornified parts of the epidermis containing the exudate.

The organism causing scarlet fever, as O. Heubner emphasizes, may enter the body through small solutions of continuity of the external skin, and lead to a general infection with localization on the surface of the skin and the mucous membranes. The stage of incubation is five days, the prodromal stage, one to two days, usually beginning with a high fever, (see under angina). The stage of eruption begins on the first or second day of the disease. The exanthem reaches its height on the second to the fourth day of the disease, and pales over the course of two to four days, after which the skin appears yellow brown. Desquamation is usually lamellated, begins at the end of the first to the beginning of the second week and is completed, as a rule, in fourteen days. The entire duration of the disease from the stage of prodromes to the ending of the stage of

desquamation, is from two to three to five weeks. The stage of infectivity is greatest during the prodromal stage, but occasionally may last from forty to eighty days after desquamation. Atypical forms of this disease are scarletina papulosa, which may become vesiculosa. In scarlet hemorrhagica s. septica, because a septic infection is added, there occur hemorrhages into the scarlet red situations, from the size of a dollar to a dinner plate; at the same time furuncles, small abscesses, and more rarely a pemphigus-like eruption may appear.

Among the complications and diseases following scarlet fever, (see under pharyngeal diphtheria p. 613, scarlatinal nephritis p. 1338), and often occurring after mild cases, usually during the third week, may be mentioned: endocarditis, lymphadenitis, particularly cervical, more rarely, inflammations of the joints (suppurative or the so-called scarlatinal rheumatism, which most frequently involves the joints of the hand and the fingers, O. Heubner), thyroiditis and others. For symmetrical gangrene, see under the chapter, gangrene. Recovery from scarlet fever practically always, though not in every case, confers immunity, but cases have been reported in which the second attack of the disease was particularly severe (Weissenberg). See Degkwitz for artificial immunization with the serum of convalescents. The cause of scarlet fever is unknown, but it has been possible to transfer the disease to apes (Bernhardt); the virus passes through a Berkfeldt filter. That the streptococcus which was found constantly by A. Baginsky in scarlet fever, is its cause, is quite improbable. Mallory described protozoa-like bodies which he found in the skin in four cases; see also Gamaleia. Field could not confirm these findings. In the kidneys of individuals with scarlet fever, and in the lymph nodes of scarlet fever material, (monkeys), Bernhardt saw intracellular inclusions similar to trachoma bodies (Hofer). O. Heubner believes that spirochetes may be responsible, because of the fact that positive Wassermann reactions without syphilis are so frequently found, especially when a scarlet fever nephritis is present (Leede), and also because of the favorable influence of the injection of salvarsan. Doehle first called attention to bodies found in the leucocytes, which stain with Leoffler's methylene blue and other dyes as globular, punctate or thready inclusions; they may, however, be found in many other febrile and infectious diseases also (Rehder, lit.), but not as regularly as in fresh typical scarlet fever. Therefore, according to the consensus of opinion, even though they are not pathognomonic of scarlet fever, they do help in the differential diagnosis of many eruptions, as for example, sepsis, angina and influenza (Kretschmar, Nicoll and Williams, Vogt, Schwenke, Isenschmid and Schemensky). Perhaps these inclusions are merely condensations in the protoplasm caused by infectious noxae; Cummins believes that they probably originate from the nucleus.

Translator's Remarks. There are dissenting opinions in regard to a streptococcus as the cause of scarlet fever, and differences of opinion in regard to the skin test, antitoxin, immunization, etc.; from the literature it is quite apparent that more time is needed for final judgment. See the work of Dick, G. F., and Dick, G. H., for example, J. A. M. A. 82; 301: 1924; 82: 265: 1924; 82: 1246: 1924, with their references, and see comparison with Italian School by Pollitzer, R., Presse Medicale 32: 1034: 1924.

(*b*) One of the mildest inflammations in the form of a congestive hyperemia has been given the name of **erythema exsudativum multiforme.** There appear red, flat or slightly prominent spots which pale on pressure with the finger, but which may occasionally be combined with hemorrhages. It is quite different from erythema volatile s. fugax which is caused only by hyperemia. There are many forms of erythema; it will suffice to name them, such as E. annulare, E. gyratum, E. iris (a new spot appears in the center of a larger old spot), E. papulosum, E. urticatum, E. vesiculosum, E. bullosum. Particular forms of vesicular erythema are herpes circinnatus, (a circle of vesicles, grouped around a center undergoing regression), and herpes iris, a ring of vesicles surrounding a central vesicle.

Etiologically, there may be considered: (*a*) the most varying kinds of external irritants, among mechanical, thermal, chemical sorts, which give rise to erythema traumaticum. As examples of these groups may be mentioned, the rubbing of clothing, the action of high or low temperatures, the action of croton oil, cantharides and gray ointment. The erythema of mercury is often spotted and localized particularly on the trunk; less often it is universal and accompanied by fever and the symptoms of mercury poisoning (stomatitis), in which it consists of thickly grouped, small, highly red, stipples, which may be mistaken for the rash of scarlet fever; the course is severe and may lead to death; v. Zumbusch believes it is possible that a sepsis is present (portal of entry, the mouth cavity?). (*b*) Many erythemas follow the administration of medicaments such as quinine, antipyrin, salicyl, chloral hydrate and others. It also occurs in pellagra (p. 2003), (Vollmer, v. Neusser), in uremia, in which perhaps retained urinous substances irritate the skin (Gruber, lit.). (*c*) Other erythemas arise from hematogenous routes during the course of infectious diseases such as pyemia, typhoid fever, in which it is spoken of as roseola, or endocarditis, and bacteria may be found in the spots (Jadassohn); (*d*) erythemas also occur in diseases of the central nervous system as well as in disturbances of digestion, particularly in children. (*e*) There also occurs an erythema exsudativa multiforme idiopathica which possibly has its foundation in a nephritis (Sachs).

Erythema nodosum s. dermatitis contusiformis s. urticaria tuberosa is characterized by an acute origin, and the appearance of boils and nodules of the size of peas to fists, of rounded, moderately prominent, rough contours, with a rose red periphery and a blue red center, or by larger, flat, or somewhat prominent spots of red color. In a few days these nodules may disappear from resorption of the infiltrates and from metamorphosis of the coloring matter, similar to that occurring in hemorrhages. Occasionally, however, abscess formation or gangrene supervenes. The etiology is not uniform: the disease may appear independently, sometimes as a complication of infectious diseases, for instance, articular rheumatism, scarlet fever, measles, pyemia, typhoid fever. According to Ernberg, the development of the disease is a signal of danger of tuberculosis, since, after disappearance of the eruption, this disease manifests itself in various ways. Many postulate an embolic origin (Hoffmann), and according to Brian, the throat is the original portal of entry in many cases. Localization is chiefly the thigh and the back of the foot, less frequently the forearm, the upper arm, the nates. Simultaneously in the same place or in other parts of the body there may appear the spots of an erythema exsudativ. multiforme. In the first eight to fourteen days new nodules appear and the disease lasts from three to six weeks. Pain in the joints and bones is often concomitant. Fever is practically always present, especially in the more severe cases.

Purpura rheumatica (p. 2072), shows a number of similarities to erythema nodosum.

See p. 160 for erythema nodosum syphiliticum. See Planner and Remenovsky concerning the benign, but painful, acute ulcers of the vulva in erythema nodosum; this condition resembles soft chancre.

In **erythema induratum** (Bazin), which is more frequent in girls or young women, there appear hard nodules of a red to a blue red color from the size of cherries to walnuts in the corium of the thigh, which after rupture (rare), empty a yellow content. Histologically, perivascular cell infiltrates and atrophy of the subcutaneous fat tissue are found in the neighborhood of the sweat glands. The finding of epithelioid and giant cells in tubercle-like foci has lead many to express the opinion that at least some of the cases are tuberculosis of the skin (erythema induratum scrofulosorum); a certain confirmation is given to this idea by the interesting experiments of Gougerot and Laroche who produced tuberculids of the skin; in isolated cases, tubercle bacilli have been found (Hidaka, lit.). Brown spots remain after healing. (Kraus, Alexander, Hirsch, Schidachi, lit., Kuznitzky.)

(*c*) **Urticaria.**—This consists of the formation of markedly itching, and burning wheals from the size of finger-nails upward, with a rosy red or a

shimmering white, slightly raised appearance surrounded by a red zone; they appear and disappear rapidly without leaving any trace. Chronic urticaria with repeated outbreaks of the urticarial rash (urticaria perstans) is called cnidosis.

In urticaria there is found a circumscribed edema (urticarial edema is also spoken of), established in the middle and upper layers of the cutis.

From marked serous infiltration of the upper layer of the papillary bodies following compression of the vessels, the plateau may be shimmering white (urticaria porcellanea). Urticaria vesiculosa and papulosa consist of wheals formed by the accumulation of fluid, or by the intravascular accumulation and wandering of leucocytes (Jadassohn and Rothe) into vesicles or bullae, or into serous nodules. These wheals may also appear in the mucous membranes of the upper air passages. From all the other kinds of urticaria there must be differentiated urticaria pigmentosa, a rare congenital affection in which practically the entire body becomes covered with brown red and yellow spots (urticaria xanthelasmoidea). They disappear only after decades, whereas urticarial eruptions usually cease after one to two years. Histologically, pigmentations of the epithelium and foci composed almost exclusively of perivascular mast cells are found in these spots (Raab).

Causes.—Urticaria arises (1) exogenously, from direct external irritation of the skin from fleas, bedbugs, lice, caterpillars, flies, cow itch (Kreibich), as well as from pollen in many individuals, not alone by primula obconica, but also from various other kinds of pollen. There is reflex irritation of the vaso-motor nerves, in part from actual penetration, and in part from itching, which may lead to the formation of wheals in situations other than the directly irritated parts. Thus, a single flea bite may produce an extensive urticaria. The rubbing of certain substances into the skin may produce typical urticaria experimentally, as for example, ethylenglycol (Baum). Occasionally, urticaria is called forth in a few seconds even after a mild touch with the finger nail; letters of the alphabet may be traced on the skin with the finger nail, and wheals soon appear in the corresponding shapes (urticaria facticia). Urticaria may also be (2) endogenous or symptomatic from irritation of the nerves of taste and those of the gastrointestinal tract. Among the foods responsible for it, there may be mentioned strawberries, crabs, oysters, caviar, fish, pork (specific hypersusceptibility, anaphylaxis), strange albumens (Wolff-Eisner, Bruck, Gluck), champagne, absynth, medicaments such as quinine, antipyrin (Zieler, Kaposi). Other, already present, diseases of the skin, marasmus (pururitus senilis), various gastric conditions, even psychic conditions, disturbances in the genital organs such as dysmenorrhea, (Matzenauer and Polland), may produce urticaria. According to Philippson, Torok

and Vas, and the experiments of Torok and Hari, urticaria is probably an inflammatory edema called forth by certain chemical bodies, following direct activity on the blood vessels of the cutis. Drug exanthemata may appear in the form of erythemas, roseolas, papules, wheals, vesicles, hemorrhages, from the most varying kinds of chemicals such as digitalis, morphine, quinine, salicyl, antipyrin, arsenic, calomel, gray ointment (p. 2078). See Jadassohn and Rothe, concerning the differentiation of urticaria and dermatitis.

Dermatitis Combustionis and Congleationis (Burns and Frostbites). Dermatitis Combustionis (Burns).—According to the intensity, there may be differentiated: first degree by temperatures of thirty-five to forty degrees Ramier, and also by the heat of the sun. There are active hyperemia with subsequent paresis of the blood vessels, and passive overfilling with blood. Moderate swelling and yellow discoloration point to mild exudation. In second degree burns, the epithelium is killed in its upper layers or throughout its entire thickness; large amounts of exudate pour from the papillary bodies which may also be injured, and the epithelium is lifted from the surface in funnel shaped, or simple, vesicles or blisters. The covering may be entirely lifted off. Drying, or suppuration of the corium follows. A scar is formed only where the papillae have been destroyed by pus (secondary infection). In burns of the third degree, the cutis is directly destroyed. In scalding, the skin is cooked, white and vesicular, or there is a swarthy drying, or actual carbonization. Dark brown arborescent figures are found in the yellow to brown leathery scabs which represent the carbonized content of superficial blood vessels. It is of medico-legal importance, that this injection of the vessels is not seen when the skin of a cadaver (with the exception of situations showing livor mortis) is burned, (E. Hofmann). See Reuter (lit.) Westenhoffer and Sury, concerning the criteria of ante and post mortem burning. In vital as well as in post mortem burning, the cadavers, following the rigor of the warmth, take on a peculiar attitude; the legs are flexed on the knees, the arms raised up to forty-five degrees, flexed at the elbows, the hands are in position of adduction; the position is spoken of as "beggar's" position. The author has seen examples of this quite frequently in death from burning gasoline, or following explosions of gasoline (see Pick, "burned aviators"). Healing occurs by granulation tissue and scar formation after the necrotic parts are sloughed off; the scars of burns are angular, ray-like, pulled in, tumor-like, lead pencil-like, or as though knitted.

There are various kinds of blisters. Some are simple vesicles which appear purely mechanically from the pressure of fluid in the corium; others are vesicles of softening, filled with the products of degeneration

of the epithelium. There may also be differentiated (Weidenfeld)' ordinary blisters of burns, in which the reddened, painful corium comprises the base of the vesicle (second degree burn), and those in which the cutis under the vesicle is white, painless and scabby; blisters of the latter kind always heal with the formation of scars.

Sudden deaths or deaths in burns which are fatal within twenty-four hours, or after about one-third of the surface of the body is burned, even though mildly, or after one eighth to one sixth of the body surface is burned to the third degree (Weidenfeld), are caused, according to the opinion of many, by alterations in the blood. These are: destruction of red blood corpuscles (Wertheim), which leads to a hemoglobinemia and a spodogenous splenic tumor (toxic), and to multiple thrombi, particularly in the vessels of the lungs (Welti, Silbermann, Markusfeld-Steinhaus, Salvioli and others). Otherwise at autopsy, degenerative changes are found in the kidneys, the heart, the liver and the stomach. In some cases, fat embolism is found in vital organs, particularly the lungs (Foa, Carrara and others). Foa even found emboli of bone marrow giant cells. He believes the influence on the nervous system is frequently the most important cause of death. Undoubtedly, the shock caused by thermic irritation to the nerve endings of the skin, brings about some sudden deaths following burns (Kucharski). Weidenfeld (lit.), believes that the nervous shock, and the changes in the blood from the heat, as well as the loss of serum, particularly in burns of the second degree (Wilms), are important, but he also believes, as does Scagliosi, that an intoxication is produced by materials coming from the burned tissues, or, as Wilms expresses it, metabolism is damaged by the products of destruction of protein; this is the most frequent cause of death, and may operate even some time after the actual burn. Neuda considers early death the result of paralysis of the heart and vessels from a toxemia, which reminds him of the activity of muscarine on the isolated frog heart (the antidote is atropine). The most dangerous form of burn in this respect is the scald. Dohrn calls attention to the similarity of an early fatal burning to an intoxication, and Wilms found a typical fever curve, which he believed due to the absorption of fever-producing substances. See also p. 1282, hemorrhages into the adrenals, which were believed by Kolisko to demonstrate the toxin theory; the toxins produced from the burned tissues damage the adrenals, and thus lead to abolition of adrenal function (diminution in lipoids, p. 1279). Most of the newer works (Pfeiffer, Kotzareff), give more or less evidence for the toxin theory, even though Ferrarini (lit.), declares that the nature of these poisons is entirely unknown. Helsted (lit.), again lays most of the emphasis on the blood (lit. in appendix). According to Birch-Hirschfeld, duodenal ulcers which are often multiple occur in about 20% of the

cases which die a few weeks after the burn (lit. by Parascandolo); the experiences of Marchand and those of the author do not confirm these figures. In late death following burns in which the larger air passages have been damaged by the aspiration of burning gases, there may be found, among other things, bronchopneumonia with a tendency to suppuration (B. Fischer and Goldschmid). See Pick (lit.), concerning the war pathology of burning, in which most of the cases were burning from a flame.

In lightning stroke, burns are found at the situation of entrance and exit of the lightning, and along its course in the skin, since the skin conducts the current better than the mass of the body. At the point of entrance are deep depressions of rounded contour under the holes burned in the clothing, but the peculiar branched, lightning figures of the skin which do not follow the distribution of the blood vessels, are believed to be partly vasomotor phenomena, and partly due to direct conduction of the current in the skin. At the point of exit of the lightning, the skin frequently shows a number of small holes with carbonized edges. Extravasations of blood into the skin also occur. In addition to the burning of the skin, there is added a mechanical tearing action on the clothing, tissues, even internal organs, and an electrical concussion; the latter is the most important, and in about 41% of cases leads to death if lightning strikes the head. See Jellinek concerning the activities of electric currents, which are quite similar to those of lightning, in producing changes in the skin which are called contact marks. Kawamura describes rounded, centrally depressed, tough, white marks in the skin at which the upper cell layers of the epidermis are pressed together and made homogeneous, while in the deeper layers, irregular cavity formation, thready appearance and basket-like arrangement of the basal cells can be seen.

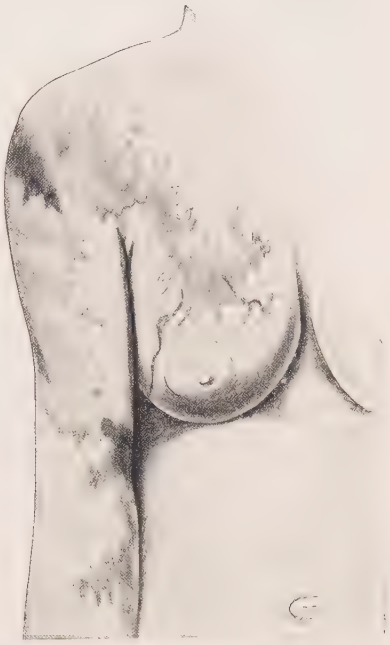


FIG. 1002.—Lightning burns on a 7 months pregnant woman 28 years old. The woman lived. (Illustr. in I. Diss, Uber Blitzverletzungen by Moritz Ebertz, Tubingen, 1891.)

X-ray burns which occur after long, or too frequent radiation, produce acute and chronic changes, manifested, often suddenly, after a latent period of about a week. The acute changes consist of erythema, vesicle forma-

tion, necrosis with subsequent ulceration as in third degree burns, and in the mildest form, depulation, (the hair grows in from six to eight weeks). Chronic changes consist of a marked dystrophy of the skin (Kienbock), an x-ray dermatitis, which is manifested by falling out of the hair, atrophy of the glands, abnormal pigmentation in the cutis, brittleness of the nails, and painful ulcerations. Carcinoma may appear in an x-ray dermatitis, occasionally in multiple situations, (see illustrations of Porter and White, Porter, and histology by Wolbach; see also Coenen). Consult the chapter, carcinoma. After prolonged treatment of lupus with Finsen light, there are hyperemia and exudation with vesicle formation and the emigration of leucocytes; after repeated applications, the lupus tissue, which is electively sensitive, is destroyed and the situation cicatrizes (Doutrelepont). See Schlasberg concerning the treatment of skin carcinoma with Finsen light.

After a transient reddening, the rays of radium produce a dystrophic necrobiotic process, leading to the formation of ulcers (Goldberg and London, Guyot, lit., Grasnick, Kehrer). See, among others, Exner, (lit.), Kehrer (lit.) concerning the treatment of carcinoma with radium, but carcinoma may follow radium treatment. See Kaiserling concerning the action of radium.

Dermatitis Congelationis (Frost-bite).—There may be differentiated, as in burns, three stages, namely, erythematous inflammation, the formation of vesicles, and necroses brought about by coagulation of the blood. Freezing of the first degree appears usually as frost-bites, a circumscribed inflammatory swelling of the skin, particularly of the hands, the feet and ears. Anemic individuals are more susceptible. Vesicles and torpid ulcerations frequently follow freezing of the second degree. In third degree freezing, the parts which are covered with vesicles or are only pale blue and marbled, undergo necrosis to irregular depths (mumificatio congelationis).

The process is due partly to "freezing" of the parts and partly to spastic ischemic gangrene (Marchand) or paralysis of the vessels, which is followed by stasis and the formation of thrombi (Wieting); in other cases it is merely spastic narrowing of the vessels with the formation of thrombi; even months after the action of cold, thrombi are wide spread and the so-called late gangrene may ensue, (Nagelsbach, lit.). It is possible for gangrene to develop even though the degree of cold is above that of freezing of water.

Later demarcation, usually by suppuration, occurs in the soft parts as well as in the bones; it often spreads into the joints; peripheral parts, such as isolated phalanges of the toes and fingers, the ears, the nose, or entire members may be lost.

For the histological details, see Kriege, Rischpler, Uschinsky, Marchand and especially Hodara for frost-bites. See Pick (lit.) for freezing as a war disease. See Zuckerkandl for illustrations of the various forms of freezing.

Sepsis may lead to death.—If the body is exposed to intensive cold for long periods of time, as for instance, a drunkard lying out all night in the gutter, all the vital functions are so depressed that death follows. If persons stiff with cold are brought quickly into a warm room, sudden death may result.

FIG. 1003.—Gangrene on end phalanges of four fingers due to freezing; female 47 years.

Miliaria.—According to Kaposi, three forms may be differentiated: miliaria rubra consists of small vesicles with red bases as an acute process; in both miliaria rubra and alba the content of the vesicles is cloudy and opalescent. According to Hebra, it is an exanthem produced by the sweat, and therefore an eczema sudamina. But, according to Unna, the contents of the vesicles have nothing to do with acid sweat, and are rather an alkaline serous exudate. Involution follows mild desquamation of the covering of the vesicles. Miliaria crystallina, or, according to Unna, merely crystallina, is a peculiar exanthem of the skin consisting of efflorescences as large as grains of corn and similar to drops of tar, which appear in large numbers, especially on the trunk (the breast and lower

trunk). They are actual sweat cysts, vesicle formations of the horny layer, with quite obvious relationship to the sweat gland pores. Healing follows by regeneration of the epithelium after the covering of the vesicles has been washed off by the sweat. Desquamation may follow. Miliaria itches, but crystallina does not.

According to Hebra, the disease is an expression of a metastatic process, since it appears frequently during the course of infectious diseases such as typhoid fever, puerperal fever, articular rheumatism, endocarditis and the acute exanthemata.

Epidemic miliaria is a rare disease characterized by marked sweating, the formation of vesicles and high fever; etiologically it is obscure, it appears in epidemic form, and is perhaps of infectious origin (lit. by Immermann, Scholtz, Vignol, Weichselbaum, lit.).

Herpes.—According to Willan, herpes is an acute cyclic skin disease, characterized by the formation of groups of transparent vesicles filled with a water-clear fluid, arranged in largest part along the course of particular cutaneous nerves. At first there appear small nodules, from which vesicles develop quite rapidly; the contents are cloudy and consist of serum, fibrin and pus cells. After hours, or a few days, the vesicles desiccate; there appear scabs; when the epithelium is renewed beneath them, the scab is pushed off. According to the localization and genesis, there may be differentiated:

Herpes labialis s. facialis on the mouth, the lips and the nose: it occurs particularly in febrile diseases such as pneumonia, more rarely in typhoid fever and cerebrospinal meningitis, etc.

Herpes praeputialis or progenitalis appears on the foreskin, the glans penis, and on the labia.

Herpes iris and circinnatus, are forms of erythema exsudativum multiforme (p. 2078).

Herpes zoster, by far the most important form, consists of numerous groups of nodules surrounded by small red rings, which quickly change into vesicles, following the course of certain cutaneous nerves, and appearing usually only on one half of the body, on the trunk, the head, or the extremities; after a number of days the vesicles dry out. From the time of v. Barensprung and more recently, the disease, which is accompanied by violent neuralgic pains, has often been considered a disease of the corresponding spinal ganglia which are the sites of inflammations extending from neighboring organs, *e.g.*, caries of the vertebral column, and, since zoster is frequent during a poliomyelitis epidemic, it is supposed that there is a localization of the virus in the spinal ganglia (Garrow, v. Zumbusch); the inflammatory irritation of the sensory ganglia produces the neuralgia. A further explanation has been brought forward, namely, that

it arises through diseases of the peripheral nerves, such as inflammatory foci in their neighborhood as from trauma, gun shot wounds, crushes, fractures or tumors (Wohlwill), as well as through diseases of the brain and spinal cord, as *e.g.*, cerebrospinal meningitis, and, more rarely, tabes (Nieuwenhuijse, lit.). It is assumed that the skin disease, the eruption of vesicles, is produced through the agency of sympathetic nerve fibers. Others believe, on the other hand, that the cutaneous arteries determine the spread of the vesicles, and that the nerves are involved only secondarily (Pfeiffer, v. Wasielewski, lit.). The term "reflex zoster" is also used when it occurs in the nerve and skin zone corresponding to that of a diseased internal organ; the carriers in this case would be sympathetic fibers which come from a diseased organ, *e.g.*, a kidney, enter the spinal cord, and from there run peripherally through a particular segment in a spinal ganglion; an irritation which reaches this path may finally produce a zoster (Hedinger). In the etiology there have been considered, furthermore: intoxications with carbon monoxide and arsenic, infectious diseases such as pyemia, intermittent fever, recurrent fever, etc. Nobl and Ullmann believe that the rare herpes zoster gangraenosus generalisatus is due to an infection. See Baum, and Luger and Lauda (lit.) for the question of the transmissibility of herpes zoster to rabbits. Other cases of zoster are "idiopathic," occasionally in epidemic form, and are sometimes contagious. For its relation to acute poliomyelitis, see p. 1990 and Lit. in appendix.

Pemphigus.—By this name are understood eruptions of varying etiology, in which vesicles of the size of lentils to goose eggs appear from unknown, but perhaps infectious, causes. The vesicles, which may be differentiated from other vesicles (herpes, eczema), by their very superficial situation, are caused by the accumulation of exudate in the epidermis. At first they are multilocular, but soon become unilocular. Their content is clear, watery, or more viscid; later it becomes cloudy from the addition of fibrin detritus, epithelial cells and leucocytes; occasionally, it is hemorrhagic. The fate of the vesicles varies. In many cases they dry and shrink; scabs are formed under which the epithelium is regenerated; no scars remain, but merely transient pigmentation (pemphigus vulgaris).

In other cases, the covering disappears, leaving an erosion, the base of which is formed by the deeper rete cells. This is frequent in pemphigus vulgaris; but regeneration readily occurs. In still other cases, the vesicles are flaccid and contain but a small amount of fluid; at their bases, in contrast to pemphigus vulgaris, there is no epithelial replacement on the corium. If the corium is exposed, it becomes covered with scabs (pemphigus foliaceus). If single layers of the corium undergo necrosis, it becomes the so-called pemphigus diphthericus or pemphigus cachecticus.

If gland-like, easily disintegrated, or secreting tender growths appear on the exposed corium, the name pemphigus papillaris vegetans is applied (Neumann). The finding of bacteria in the vesicles (Demme and others), does not as yet permit any definite etiological interpretation; but lately many authors lean toward the infectious theory (lit. appendix).

Clinically, various forms of acute and chronic pemphigus may be differentiated:

Acute or febrile pemphigus; according to Kaposi, this occurs chiefly in children, occasionally in epidemic form, with or without fever. Vesicles from the size of peas to beans appear, distributed irregularly on the face, trunk and extremities. After several hours or days, they dry, form scabs and leave pigmented spots. Desquamation takes place inside of eight to ten days. This form is known as pemphigus contagiosus infantum.

Pemphigus syphiliticus occurs chiefly in the new born (see section on lues); more rarely it appears in adults, *e.g.*, over ulcerated nodules.

Pemphigus neonatorum simplex congenitus is relatively rare and probably of infectious origin (Richter, Labhardt and Wallart). According to Reinhardt (lit.) this type is closely related to dermatitis exfoliativa (p. 2089), and differs only in degree. It appears in the new born during the first weeks of life, as unilateral vesicles without typical situation; they may reach the size of dinner plates. Various microbes in large numbers have been found in the contents, and, according to Lipschutz, they are somehow related to the Leishman bodies (p. 236).

Pemphigus Chronicus Vulgaris.—Eruptions of vesicles appear periodically in the skin and the border of mucous membranes to the accompaniment of fever. It occurs both in children and adults. Eosinophilia can be found in the blood (p. 197). Occasionally the grouping of the vesicles leads to a particular designation such as pemphigus disseminatus, circinnatus, gyratus, serpiginosus, etc. Pemphigus occasionally takes on a diphtheritic character. The course: non-complicated pemphigus vulgaris often ends in from two to six months, with a long drawn out convalescence, but later, new eruptions may appear. Malignant forms, which are usually fatal, occur in violent relapses producing cachexia, or they change into pemphigus foliaceus, pemphigus diphthericus, or pemphigus papillomatous.

Pemphigus Foliaceus.—The relaxed vesicles become larger, closer together, spontaneously or mechanically lose their covering and leave the corium exposed. The places become moist, and are covered with varnish-like crusts. In months to years, the entire surface of the body may be covered by these structures when the individuals become marantic, and practically always succumb. Bullous efflorescences appear in the mucous membranes of the upper air passages, and occasionally in the conjunctivae.

Pemphigus papillaris vegetans is the most malignant form. There appear groups of vesicles the size of lentils or larger, with new eruptions around their edges; the centers sink after the vesicles rupture, and become covered with crusts. Then the former base of the vesicle begins to grow, and tender condylomatous papillary growths produce a gray discolored wound surface (differentiated from syphilitic papules, see illust. by Rille). In the most malignant types which end fatally in a few months, the process proceeds in a serpiginous form; the edges are always vesicular and raised. Since the mucous membranes of the mouth and the pharynx are attacked first, mistakes for syphilis are easily made. In the mouth, the efflorescences last many months as erosions or aphthous ulcers. If this variety is implanted upon a pemphigus vulgaris, particularly the circinnate variety, healing is still possible (lit. by Wiegmann).

Pemphigus neuroticus is the name given to cases which occasionally follow, or appear during the course of diseases of the nervous system. The findings in the spinal cord and the sympathetic system have varied considerably (Schwimmer and others); pemphigus-like eruptions in an older myelitis, vesicles in neuritis, and pemphigus leprosus, are usually given as examples. But the apparent relationship of the vesicular eruptions found in the skin and central nervous diseases, does not necessarily uphold the neuropathic origin of typical pemphigus (Schlesinger). (Lit. concerning pemphigus in the appendix.)

Hereditary epidermolysis bullosa (Kobner) appears in isolated cases, even before birth, (dermatitis exfoliativa neonatorum), but usually after the child begins to walk, in the form of vesicles between the corium and the epidermis, occurring in certain situations from purely mechanical, mild, but long lasting friction traumata. For this reason, Sakaguchi prefers the name epidermolysis bullosa hereditaria traumatica. The contents of the vesicles are yellow, clear, or hemorrhagic. Heredity has been determined in half of the cases. The male sex is preferred. It is usually assumed that there is an abnormal reaction of the cutaneous vessels to mechanical stimuli (angioneurosis), which leads to increased secretion of lymph; according to Polland, this disease is related to acute cutaneous edema (Quincke, p. 2070), and to urticaria. Stuhmer believes the explanation lies in a disturbed balance between the thyroid and the vasomotor system, whereby an abnormal irritation is exerted on the nerve centers which regulate the vasomotors. Literature by Luithlen and Berger, Sakaguchi, Beck, Zweig.

Eczema.—Eczema is a very common, clinically indefinite, skin disease which runs an acute or chronic course.

It is caused chiefly by external irritants of various kinds such as medicaments,—mercury, carbolic acid, iodoform, terpentine and others; thermic

influences (eczema solare in mountain climbers); mechanical causes, and occasionally infections, *e.g.*, tubercle bacilli in the eczema of scrofulous individuals. Disturbances in the circulation of the skin favor its appearance *e.g.*, eczema cruris in varices (p. 167). Many cases are obscure in their etiology (internal causes?). In certain families, the affection is hereditary. For the typical course of an eczema, especially the acute stages and the exacerbations, Scholtz believes that a staphylococcus infection is constant and causative (Jadassohn, Unna, Veiel, Bruck and Hidaka).

According to Kaposi, eczema begins with punctiform or diffuse swelling and reddening of the skin (eczema erythematosum), or with itching nodules (eczema papulosum). It may then disappear or progress to vesicles (eczema vesiculosum), and following rupture of the vesicles spontaneously or after scratching, a serous or a seropurulent fluid appears on the surface, a wet eczema (eczema madidans). Following this, crusts form on the surface from drying of the exudate (eczema crustosum). Eczema impetiginosum occurs when a sero-purulent secretion accumulates under the crusts. Recently, this form has been differentiated into a separate disease, namely, impetigo, (see below). If the secretion ceases, the skin under the crust becomes covered with a layer of epidermis; the crusts are pushed off, exposing a reddened and shimmering skin (eczema squamosum).

Course.—Restitutio ad integrum may occur, often with brown pigmentation remaining, or recurrences occur from which a chronic eczema may be developed. The histological changes in acute eczema are like those of erythema papulosum and herpes.

The chronic form always leads to spotted, striped pigmentations, thickening of the epidermis and corium, whereby the surface becomes covered with trabeculae, scales or plaques of epithelium. More marked hypertrophy of the papillary bodies produces an irregular, warty surface. Higher degrees of hypertrophy of the connective tissue of the cutis and subcutis as well as of the epithelium produce changes which are similar to those of elephantiasis arabum. In the cutis, the connective tissue of which becomes sclerosed, there are dilated blood and lymph vessels, destruction of the hair follicles and of the sebaceous and sweat glands. In the subcutis, the fat tissue is compromised by cicatrizing connective tissue. All of these changes are seen most frequently in leg ulcers (p. 167).

Chronic eczema of the scalp produced by head lice or associated with them (pediculosis capitis), may lead to complete adhesion of the hair in women and forming of "felt" (plica polonica).

Erythema intertrigo, consisting of diffuse reddening, and the watery eczema intertrigo produced after removal of the epidermis, are particular

forms of eczema, which appear in the genital folds, under the breasts, and are similar to the condition brought about by mycoses (Kumer, lit., and p. 1803). Serious ulcerative destruction may occur in sucklings.

Impetigo. Ecthyma.—Impetigo was formerly known as the impetiginous form of eczema, but recently has been regarded as an independent disease. There are differentiated:

(*a*) **Impetigo staphylogenes** (Unna), related to the follicles, begins as small pustules, leading to follicular (folliculitis), and perifollicular (perifolliculitis) pustules, which later change into dirty crusts. Sycosis of the beard is regarded as a perifolliculitis. From a perifolliculitis, further invasion of the staphylococci into the tissues may produce furuncles and carbuncles. It occurs in the face (region of the beard), hands, and arms in adults.

(*b*) **True impetigo contagiosa** (impetigo vulgaris, Unna) begins as a small, usually flat, relaxed, vesicle about as large as a pea, containing a clear or mildly cloudy content. When the vesicle bursts, the exudate coagulates, and forms a transparent honey-colored crust. The vesicular eruption may proceed around the peripheries, but the crusts predominate. After removal of the crust, healing takes place leaving a red surface. According to Unna, it is caused by invasion of staphylococci along the hair follicles and sheaths, into and between the horny layers and those below the epidermis; type (*a*) appears when they penetrate deep into the shaft of the lanugo hair and its sheath. It occurs in the face and hands, chiefly in children. The disease is readily transmitted from one child to another. Etiologically, it is mainly an infection of the superficial skin with either staphylococci and streptococci, or both (Scholtz). Lewandowsky believes that a symbiosis of both kinds of cocci is the most frequent finding. See Eug. Fraenkel, (lit.) concerning hematogenous metastatic dermatoses during the course of septic diseases.

Ecthyma is a more severe form of impetigo; the pustules are larger, the infiltration more extensive. Dirty ulcers appear when the covering of the pustules has been removed and their contents dry. During the war, it was seen most frequently in the thighs, feet and forearm. It was usually accompanied by other "pyodermias," such as infections of the skin with ordinary pyogenic organisms (Jadassohn), furuncles, abscesses, etc. They show a poor healing tendency (Kromayer). See Takahashi (lit.), concerning eczema gangraenosum during the course of measles; he believes that there has been a hypersusceptibility (anaphylaxis), of the skin to bacterial infection arising during the course of the measles. For eczema gangraenosum in pyocyaneus infection, see p. 1763.

Small Pox.—According to Kaposi, small pox is an acute contagious disease, characterized by fever and other general symptoms, and the

appearance upon the entire skin of an eruption of papules, vesicles and pustules, all of which run a typical course. The disease is a result of infection of the blood with the specific virus.

The most likely portals of entry are the mucous membranes of the upper digestive and respiratory tracts. As the animal experiments of Paul have shown, these same situations are also the most important and the earliest portals of exit of the infectious virus (droplet infection; see Helly).

The vaccination law of the 8th of April, 1874 has made small pox a very rare disease in Germany.

Variola Vera.—After an incubation period of about two weeks, the prodromal stage begins very suddenly with high fever; it is accompanied occasionally, but not always, by an exanthem (roseola variolosa), which consists of a scarlet fever-like redness (erythematous form), or of punctiform hemorrhages into the cutis (hemorrhagic form). The prodromes usually last three days, whereupon the stage of eruption begins. With tenderness in the skin, there appear wildly red, conical, tough papules the size of corn grains and larger, which in isolated form on the trunk are surrounded by broad hyperemic margins. The head, the face and the parts covered by hair are usually involved first, and most richly, exceptionally, the forehead is first affected, then the trunk, the extremities, particularly their ends; the upper half of the body usually shows the disease earlier than the lower half. Those parts which have been irritated, or have been under pressure for a longer time before the disease (*e.g.*, under a truss) are frequently strewn quite thickly with the eruption (variola confluens; H. Curschmann and Risel, lit.). Sharply demarkated vesicles under tension then appear upon the prominent nodules, many of which show central depressions. This is the stadium floritionis (6th day of disease). In the beginning, the contents of the vesicles are serous and clear. On the ninth to the tenth day it becomes purulent, and the vesicle becomes a pustule as the depression disappears. The neighborhood of the pustules is intensively reddened and edematous. The entire face may be edematous, swollen and unrecognizable. The acme of the process is thus reached. The fever increases again. Papules may appear on the mucous membranes of the upper air passages. The stadium exsiccationis begins on the twelfth to the thirteenth day in the less severe cases; fever is diminished; the pustules sink, first at their points (secondary depression), and as the contents and the coverings dry, form brown flat scabs. The swelling in the neighborhood gradually disappears. Those pustules which burst here and there, particularly in the face, become covered with a yellow crust. From the sixteenth day onward, many scabs are loosened; red or brown spots remain from three to four weeks in the palms of the

hands and the soles of the feet. Otherwise, decrustation is complete by the beginning of the fourth week. Small pox usually leaves behind only a brown spot, but occasionally, (see below), there remains a white, glistening, slight depression in the skin. See Jochmann, W. Fischer, concerning the

FIG. 1004.—Suckling with small pox.

blood picture in small pox, myelocytosis, with large multi- and uninucleated cells and large lymphocytic irritation forms.

The microscopic appearance of the typical small pox efflorescence (fig. 1005), shows that its chief site is in the epidermis; under the influence

of the virus, there are swelling, cloudiness, and foamy change (coagulation necrosis) of the cells of the rete Malpighi, above the points of the papillary bodies. Exudate enters the epithelium from the vessels of the papillae as

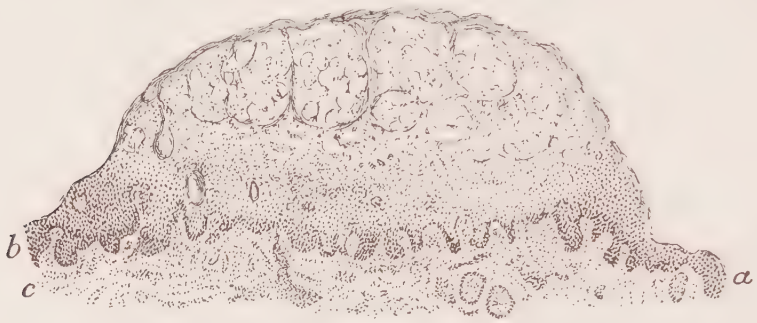


FIG. 1005.—Section through pustule of small pox; a, horny layer; b, epidermal deeper layers; c, corium and its papillae with small cell infiltration. The pock consists of a system of holes in the epidermis of which ridges remain; meshes filled with serous exudate and invading pus cells. (Magnifying glass.)

they become surrounded by round cells, and thus there appear receptacles in the swelling focus which gradually develop into an umbilicated vesicle.

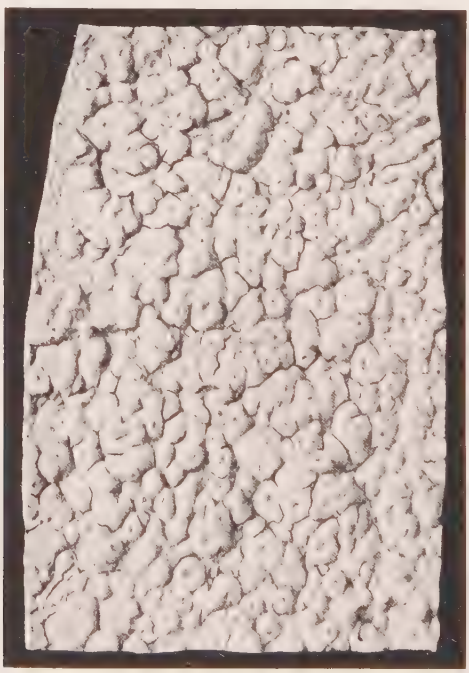


FIG. 1006.—Piece of skin with confluent small pox; nat. size.

These receptacles are composed partly of layers of degenerated cells, partly of thicker columns of Malpighian cells, and partly of misplaced

nuclei-containing epithelial cells. The dimple appears because a tougher column of epithelium remains in the center. The structure is bordered below by interpapillary rete cells and by exposed corium.

An epithelial layer difficult of destruction is necessary for the formation of a typical vesicle or pustule. No vesicles are formed in the mucous membranes (Eug. Fraenkel, Verse). The corium is infiltrated by small cells. As the vesicle becomes pustular from the accumulation of cells, the limiting walls of the multiloculated chamber gradually melt until a uniloculated pustule results. Then the content coagulates with the covering of the pustule and dries, whereby a scab is formed. The scab is loosened by the growth of epithelium from the remaining parts and the scab is gradually pushed off. In many cases of true small pox and in almost all of varicella and varioloid, the remains are only brown spots and no scars, because the papillae of the corium are not destroyed.

When, however, there is much suppuration, and the corium is destroyed from infiltration, deep, diphtheritic and gangrenous areas appear which cover the corium with a yellow necrotic mass, or extend into it and soften it. On healing, these leave typical small pox scars. They are quite frequent, particularly after variola confluens, chiefly in the face.

Atypical Types of Small Pox.—Variola hemorrhagica (black small pox), is an atypical form in the unfavorable sense (Kaposi); it usually occurs in vigorous younger individuals because of a high susceptibility (v. Pirquet); the exanthem is earlier than in variola vera, and two forms may be differentiated: (*a*) purpura variolosa, which runs the course of a severe sepsis, and shows a scarlet fever-like dark purple red discoloration, followed by hemorrhages into the skin, whereby black brown spots appear on a purple red background; they may reach considerable size in a few hours. There are exceptions, in which only small blood spots appear on the trunk. As a rule, the course is fatal in about twenty-four to thirty-six hours. At autopsy, hemorrhages are found in all the internal tissues and organs. Swelling of the spleen, however, is lacking. In a case published by Boehm lit.), among other things, there were diffuse hemorrhages into both pelves, and a blood picture which indicated a marked irritation of the myeloid system, inasmuch as there were large numbers of young bone marrow elements in the blood. (*b*) In variola hemorrhagica pustulosa, hemorrhages appear in the pustules in one to two days; the pustules begin typically with epithelial necrosis and exudation, but their cavities are filled with blood. Spots may occur as in (*a*). Death usually occurs quite early.

Among the internal organs involved with the skin exanthem may be mentioned: (1) efflorescences and necrotic processes in the mouth, the pharynx, the esophagus, the nose, larynx, trachea, vagina and rectum.

Mixed infections play an important role in these necrotic processes. There are also found (2) degenerative inflammatory processes in the myocardium (p. 61), the kidneys (p. 1339), the liver (p. 906), the testes (p. 1484). An osteomyelitis variolosa (Batzdorff, lit., p. 1096), is also common, usually in the form of very tiny foci. Not rarely, there appear (3) hemorrhages into the ovary, from the mucous membranes of the tubes and the uterus (pseudomenstruation). From secondary infection with pyogenic organisms, particularly streptococci, there occur bronchopneumonias, which not rarely lead to death, otitis media, parotitis, etc.

Varioloid is a mild form of small pox in which drying occurs in the papules before suppuration appears, and in which the efflorescences are much less in number. Such cases may appear at the time of an actual small pox epidemic. Kyrle and Morawetz obtained varioloid by intravenously injecting monkeys with the blood of those sick with small pox.

Varicella is an independent disease with histologic differences from variola pustules, in that giant cells are present (Paschen, Hammerschmidt, lit.). After a fourteen day period of incubation, and a febrile prodromal stage of twenty-four hours, water-clear thin-walled vesicles on a reddened background appear two days later. Umbilication is very often lacking. As visible traces, there may remain milk-white spots in the surrounding skin, or in the parts themselves.

Cow pox (**vaccinia**), which occurs in milk cows, almost exclusively on the udder, behaves anatomically as the efflorescence of true small pox. An original cow pox does not occur, but through vaccination from humanized vaccines by human hands. The ordinary vaccination as practiced in immunization (Jenner, 1796), by the transmission of cow pox to human beings, shows the same anatomical changes as true small pox. It occurs occasionally on the hands and axillae of milkmaids, rarely as a vaccinia generalisata. Immunity lasts about ten years. See Noguchi concerning the cultivation of vaccine virus in the testes of rabbits and their transmission from testis to testis.

The question of the actual cause of true small pox is still open (Gins, lit.). According to v. Wasielewski, the etiological meaning of the cytorycytes (Guarnieri), *i.e.*, bodies found in the epithelial cells, which are transmissible with the lymph to the cornea of rabbits, is quite possibly definite, and Councilman, Magrath and Brinckerhoff, and their co-workers, confirm completely the causative relation of these cytorycytes. On the other hand, (Huckel), these Guarnieri bodies are thought by others not to be the causative organisms, but merely a specific cell reaction to the still unknown virus of small pox. According to Hammerschmidt (lit.), the bodies are specific and characteristic of the disease, and appear from the activity of an unknown virus or its toxin on the nuclear substance;

this latter is increased, makes its exit from the nucleus into the cytoplasm, and alone, or with the help of other substances, gives rise to these bodies. In the protoplasm of cells, the nuclei of which contain rounded bodies described by Councilman and others as parasites, Paschen found heaps of just visible points which he calls "elementary bodies;" Hallenberger believes these bodies which may be found extracellular in the content of the pustule (exception Voigt, and of great diagnostic importance, E. Becker), are quite probably the cause of the disease. They seem to be identical with v. Prowazek's intracellular "initial bodies." Glins (lit.) believes that they are not a reaction product of the cells, but are independent foreign bodies, which quite probably represent one form in the development of the parasites of vaccine virus. "Inclusion bodies" as found in small pox, may be found in other chlamydozoan diseases—v. Prowazek—produced by chlamydozoa, *i.e.*, organisms on the border of the visible, which have the peculiarity of producing inclusions and specific reaction products inside of the tissue cells, as in distemper of dogs, rabies (p. 1992) chicken cholera, etc. For a good review of these cellular inclusions, see Lipschutz; for their staining see Boing. In the pus of the pustule, the ordinary pyogenic organisms may be found. Transmission is through the scabs, the sputum, and the nasal secretions of sick individuals (the latter, particularly important); dried materials retain their virulence for a very long time (in the pus of the eruption, even after years). Even in the cadaver, the virus retains its viability. In the differential diagnosis, a most important procedure is vaccination of the contents of a pustule on the cornea of a rabbit (Paul).

There also occurs a variola sine exanthemate, from which probably all transitions to complete immunity may occur (R. Massini).

(Lit. in appendix.)

Psoriasis.—Psoriasis is a chronic skin disease characterized by dry, white, glistening scales, accumulating in the form of punctiform eminences, or larger flat plaques on a sharply bordered, red, easily bleeding background. The primary exanthemata in the various forms of psoriasis are all alike, and produce brown red nodules the size of the head of a pin, which in a few days become covered with white, easily removable, epidermis scales.

If many nodules appear simultaneously, the name psoriasis punctata is applied. By peripheral spread, the areas become larger and appear like sprinkled drops of mortar, or moderately swollen plaques, covered by scales (psoriasis guttata and nummularis). By spread of the red edge, areas as large as a silver dollar may appear in a few weeks, or, by confluence of the plaques, map-like figures or diffuse areas appear—(psoriasis figurata and diffusa). If the process spreads peripherally and heals in

the center, the condition is known as psoriasis annularis, and if many such areas coalesce with the formation of snake-like lines, the term psoriasis gyrata is applied. Healing leaves normally colored, pigmented or decolorized areas. Localization: psoriasis may appear in any part of the integument. The extensor surfaces of the arms, the knees, the scalp, the sacral region are preferred. Almost always these spots are covered with thick, dirty scales. In contrast to syphilitic psoriasis, the soles of the feet, the palms of the hands, even in universal psoriasis, remain free in the great majority of cases (for exception, see Piccardi, and p. 2137). But more recent investigations seem to show that these situations are more frequently involved than was previously thought (Burgener-Jadassohn, lit.).

Microscopically, the papillary bodies and the layers of the corium immediately underneath are infiltrated by round cells, and the blood vessels are dilated. The papillae may become larger through overgrowth (J. Neumann). According to Kaposi, the disease of the papillary bodies is the primary lesion. The cells of the stratum corneum are pushed forward in the form of scales, between which air enters, whereby they become white or silvery. Normal cornification is thus disturbed (Martinotti), the cells dry, and the various cell layers lose their cohesion. (Histological details by Haslund.)

The etiology is still unknown; many postulate a parasitic cause, and believe in ectoparasites (Lang and others, and recently Raske, a spirochete), others in an endogenous origin, inasmuch as it is an infectious disease, in which the skin symptoms are merely an expression of the cutaneous projection of a dermotropic virus latent in the organism (Lipschutz, lit.). Others believe inheritable disturbances of metabolism such as gout, diabetes and others, are etiologically more important (Marcuse). Bettmann postulates a particular predisposition of the skin itself. See also Riecke. The disease is certainly hereditary (v. Hebra), but not contagious. It is said to be particularly frequent in chronic alcoholics and be subject to most intensive recurrences. Therapy (arsenic, and iodine), does not seem to cure the disease, but merely relieves isolated eruptions (Riecke, lit.).

See p. 2137 concerning psoriasis syphilitica. See Jordan (lit.) concerning psoriasis vulgaris mucosae oris which is very rare. See the special dermatological literature (comprehensive lit. by Martinotti, summary by Gudemann), concerning parapsoriasis (en gouttes, lichenoide, en plaques), under which term von Brocq gathered a number of rare skin diseases which lie between psoriasis vulgaris, pityriasis, lichen ruber planus, mycosis fungoides and eczema seborrhoicum.

Prurigo is a disease which usually shows itself during the first two years of life, and lasts the entire life of the patient, as a chronic recurring erup-

tion of markedly itching papules about the size of a pin head, which in the beginning are uncolored or pale red. Later the papules become larger or brown gum-like scabs. In its further course, the scratching produces excoriations, pustules, striped and diffuse dark brown pigmentations, and finally a thickening of the skin, particularly along the thighs, with swelling of the lymph nodes (thickened inguinal glands), draining the corresponding parts.

Chief Site.—Extensor surfaces of the extremities with increasing intensity from above downward, also on the trunk. The flexor surfaces of the knees, elbows, axillae, the inguinal region, are always free of nodules. The etiology is obscure. Many believe prurigo belongs to the neuroses.

Lupus erythematosus (Cazenave) or erytheme centrifuge (Biett), has nothing to do with true lupus. Red, raised spots appear, as large as millet seeds to lentils, the centers of which are depressed, and are covered by a dry yellow scale, or are without scale and smooth, like a scar. These spots represent a sort of primary efflorescence of lupus erythematosis (Kaposi). The nose, the cheeks, the ears, the scalp, the fingers, toes, are sites of preference of the disease.

In its further course, there then appear two chief forms:

(*a*) Lupus erythematosis discoides. As the border extends peripherally, it is the site of black comedo points, or open gland mouths, and the centers become atrophic; thus in the course of many months, there appear islands surrounded by a red area, which may reach the size of a dinner plate. After many years, the process may finally heal.

(*b*) In lupus erythematosis disseminatus aggregatus or folliclis (Barthelemy), numerous discrete foci appear, either all at once, or one following the other. As shown particularly by Boeck, the typical efflorescence consists of tough nodules in the subcutaneous tissue, as large as the head of a pin to a lentil, freely movable, and sharply outlined on all sides; this prominence then pushes the cutaneous papule onto the surface. The papule softens in the center, and this central situation (pus focus, Boecks), becomes a small encrustation, which is pushed off after a few days, and is followed by healing with a deep scar. Recurrences are frequent. Atypically, the development of nodules leads to larger, smooth or rough nodes. In rare cases, they may be distributed almost universally. Very rarely, it is acute with a universal eruption and fever, in which larger nodules are formed, and in which the end is usually death (Kaposi). See Kraus and Bohac (lit.) concerning the coincidence of pneumonia and this disease; they believe there is a direct relationship between them, and that it is a toxic angioneurosis.

The reason for the process, according to Neumann, Kaposi and others, is a degeneration and atrophy with inflammation of the entire layers of the

cutis in which all glandular parts cooperate. Just as erythema induratum, (p. 2079), and lichen scrofulosa (see p. 2101), this condition has been classed with the so-called "tuberculides," which are believed to be due to the toxin of tuberculosis (Besnier, Boeck, Roth, Alexander, Juliusberg, lit., positive vaccination experiments of Gougerot-Laroche, Zieler, Ehrmann, and Reines, Bloch and Fuchs, lit.; see also lit. and newer theories concerning the relation of lymph gland destruction and ferments reaching the blood from the destroyed lymph nodes and producing anaphylaxis by Gennerich); but, according to Pick (lit.), and Torok, this is not justified, and, according to Hidaka, is true only of (*b*). According to Bauer-Jokl (lit.), various bacteria, staphylo-, strepto-, pneumococci, influenza and tubercle bacilli, (see also Arndt), may be considered as microbic factors; a particular reaction, on the part of the skin, is said to be an obligate predisposing cause. The course is very chronic, from ten to twenty years. The end is practically always a cicatrizing change in the skin. But many spots disappear without leaving scars.

Lichen.—Previously, the term lichen was used in a general sense. Now the designation is reserved for very chronic skin diseases, in which nodules are formed, which, apart from their growth and desquamation, remain unchanged as such, and eventually undergo involution, never changing into vesicles or pustules. Different forms may be separated:

In **lichen ruber planus,** there appear flat, umbilicated, and, when scaly, light red nodules, with a smooth, wax-like glistening umbilicated covering, scattered without particular arrangement (Hebra). It is usually the extremities which are primarily involved, later the trunk. There are no scrofulous symptoms. It is quite frequent in the mucous membrane of the mouth (p. 554) and also on the nails. The etiology is obscure. For the alterations and the cornification processes, see Martinotti. In lichen ruber acuminatus (Kaposi), there are tough, brown red or pale red, rounded or pointed nodules from the size of grains of corn to the heads of pins, which, in the beginning, are particularly thick on the abdomen and breast, and then melt more and more to desquamating plateaus. During the course of years, the entire body may be involved. Nodular, tough, raw, papules appear in lichen ruber verrucosus. Histological: in both kinds of lichen ruber, the hair follicles, and particularly their neighborhoods, are the seats of the disease. In fresh efflorescences, there are dilatations of vessels, cell infiltrations, pigmentation in the corium, lack of pigment in the rete, and older hyperplasias of the stratum corneum, and the rete Malpighi. In the acuminatus, the stratum granulosum consists of seven instead of two to three cell rows, and the pigmentation and infiltration in the corium are more marked. In the verrucose variety, there are hyperkeratotic changes in the epithelium, which,

however, regress as the corium becomes infiltrated. Cystic dilatations of the sweat glands are quite frequent and numerous (Juliusberg, lit.). There is also an atrophic form of lichen ruber planus (lichen atrophique, Hallopeau), with marked regressive disappearance of tissues and scar-like changes. Darier calls this form "lichen sclereux;" it may develop from a typical lichen ruber planus (Besnier and others, Zarubin, lit.). (Lit. by Bettmann, Polland, Riecke.)

Lichen scrofulosorum, or, better scrofuloderma miliare, (Neisser), produces groups and foci of early white, later red to brown red nodules, with mild desquamation, more rarely with small pustules on their points. Localized on the back, with acne (cachecticorum), it occurs particularly, (according to some, exclusively), in chronic tuberculous individuals. Each papule corresponds to the opening of a follicle and its neighborhood; cells and exudate appear in and around the hair follicles and in the sebaceous glands. In addition to this perifollicular infiltration, there is hyperkeratosis; masses of the horny layers come from the openings of the follicles, and produce the scales. The course of the disease is benign; the papule may disappear completely. While Hallopeau and Boeck are of the opinion that it is a tuberculo-toxic condition, and that it belongs to the so called "tuberculides," Sack, Jacobi and Wolff, the two latter on the basis of finding tubercle bacilli, believe that the condition is actually a perifollicular tuberculosis. The histological picture in some of the cases, consisting of cellular nodules with epithelioid and giant cells, resembles tuberculosis or lupus. Thus, in some of the cases, the condition must be classed with the true skin tuberculoses. Klingmuller (lit.), believes that lichen scrofulosorum is an effect of toxins coming from the tubercle bacilli, without necessarily meaning that the localized appearance of the lichen eruption depends upon the presence of living or dead tubercle bacilli. Animal experiments up until now have been entirely negative. (For tuberculides, see p. 2100.)

Erysipelas.— Erysipelas is an acute inflammation of the skin caused by the streptococcus (Fehleisen), which, as a rule, spreads rapidly, and is accompanied by general febrile changes. (See Prausnitz, E. Schmidt and others for the rare cases of swine erysipelas in humans, which appears as a local inflammation in the form of red "brick dust" blisters in the region of the infection, but which may become a sepsis and lead to death. For more details concerning the bacillus erysipelatis suis, see v. Baumgarten.) The streptococcus Fehleisen is identical with the streptococcus pyogenes (see, among others, v. Baumgarten). It enters the skin through abrasions, but probably can also reach particular places from the blood stream. The injuries which act as portals of entry are usually so small that they are unnoticed.

As a complication of the healing of wounds, erysipelas is now quite rare.

As v. Recklinghausen and Lukomski first showed, the bacteria localize in the spaces between the connective tissue, and in the lymph vessels, chiefly in the most superficial layers of the corium, and possess the peculiarity of not leading to suppuration (phlegmon), but of producing a vigorous, cellular exudative inflammation of the skin and the subcutaneous tissues. Inflammatory hyperemia, cellular, serous, or cellulo-fibrinous infiltrations are established in the skin, and may spread into the subcutaneous fat tissue; to a minor degree there is also degeneration of the tissues.

The skin appears swollen, smooth, shiny, intensively red; the redness extends peripherally, often as though with pseudopodia; in the early stages it gradually disappears into the neighborhood; later it is sharply bordered. As quickly as they appear, the redness and infiltration may disappear (two to three days), and even on the fourth day, the skin may appear normal or may become brown red, dry, and desquamating. Usually the disease lasts eight to fourteen days.

Since the shaft of the hair is separated from its roots by exudate, the hairs fall out, and there is a defluvium capillorum. When the inflammatory changes are more intensive, an erysipelas vesiculosum may appear, wherein the cells of the rete Malpighi swell and undergo liquefaction. In erysipelas pustulosum, the contents of the vesicles become purulent; in erysipelas crustosum, they dry to scabs. In erysipelas gangraenosum, a part of the tissue becomes gangrenous after having been richly infiltrated by cells. Erysipelas phlegmonosum represents the transition to a phlegmon. A progressively septic erysipelas leads to death from severe general symptoms.

On rare occasions, an erysipelas extends into a relatively superficial joint (knee, ankle) where suppuration ensues, (Hildebrand).

The so-called Rosenbach's erysipeloid has its seat usually in the hand, more rarely in the face, the head, and the skin of the neck, and still more rarely in the lower extremities (Ehrmann).

Streptococci have been found in the blood of individuals with erysipelas (v. Noorden). Sites of preference are the face, from which it spreads quite readily to the scalp, the neck, and the nape of the neck. The regional lymph nodes swell. Repeated erysipelas may lead to elephantiasis and the face becomes mask-like; if in the leg, a leg ulcer may follow. Most cases of erysipelas end favorably. The inoculation of erysipelas into malignant tumors (Busch, Volkmann, Janicke and Neisser, Kolaczek), causes partial necrosis of the tissues (see also Wolffheim). Occasionally an intercurrent erysipelas seems to act favorably on gummata (Strack, Lang and others).

Phlegmon of the skin is an acute diffuse inflammation of the skin, and subcutaneous tissues, which proceeds, as a rule, with suppuration; in its early stages, it leads to more or less extensive liquefaction of the tissues (solution from ferments liberated in the pus). In situations where the skin is thin, the appearance may be that of erysipelas. Phlegmon is therefore also called pseudo-erysipelas, even though the inflammation in erysipelas is much more evanescent, and does not lead to solution of tissues. Occasionally, during the course of phlegmon there is an inflammatory, edematous swelling and lifting off of the epidermis in the form of vesicles. Because of the combination of inflammatory edema (the exudate is yellow and more viscid in comparison to the almost colorless, watery, simple, hydropic fluid), and suppuration, the term "purulent edema" is also used.

In addition to involving deeper subcutaneous parts, the inflammation may spread to neighboring tissues such as the fascias, tendon sheaths, the intermuscular tissue, and even the periosteum. Lymphangitis, thrombophlebitis or fatal pyemia are quite frequent.

Along with the tissue suppuration, there are more or less extensive necroses, and finally ichorous abscesses, in which necrotic tissue parts are embedded. The abscesses may perforate externally.

A phlegmon localized to the finger is called a panaritium, or panaritium cutaneum et subcutaneum; when the periosteum is also involved it is called panaritium periostale; when the process is situated under the nail it is called panaritium subunguale.

In the **etiology of phlegmons,** the most important and the most frequent causative organism is the streptococcus pyogenes, with the staphylococcus pyogenes aureus second. The bacteria primarily reach the tissues either through open wounds or through unimportant injuries in the skin. In pyemia, metastatic phlegmons are the rule. See p. 2042 concerning the so-called gas phlegmons without decomposition, which are produced by anerobic lactic acid bacteria, and which run their course without macroscopic suppuration. The most severe forms of putrid phlegmons are caused by the entrance of anerobic decomposition organisms with streptococci. A phlegmon may also, as in a case of Schottmuller, or as in rat-bite fever (Japan, Sodoku), run a febrile and general course and be accompanied by severe edema, or, as in erythema nodosum, with livid red spots (Martinotti).

Phlegmons without pyogenic organisms (so-called aseptic suppurations), may be caused by the subcutaneous injection of even minimal amounts of croton oil or turpentine. After snake-bites, acute edematous swellings occasionally appear in the subcutaneous tissues, which ordinarily disappear rapidly without suppuration. See Calmette, concerning snake

poison. The so-called woody phlegmon (phlegmon ligneux, Reclus), is a board-like infiltration of the neck in which the skin becomes leathery reddish violet, without, however, any tendency to softening until very late. It may be mistaken for a malignant tumor. In all probability it is due to an infectious process (Kutznetzoff, Richard).

Acne, Furuncles, Carbuncles. By acne is understood tiny papules or pustules of inflammatory origin, which arise in the neighborhood of the shaft of a hair or a sebaceous gland. Sebaceous material or pus may be expressed from the reddened papule. Occasionally the sebaceous glands or the hair follicles are destroyed by the suppuration. Acne may appear in all situations with the exception of the palms of the hands and the soles of the feet.

Varying degrees of inflammation lead to the differentiation of various forms: acne punctata, pustules with comedos in their middle; acne pustulosa, suppurative papules; acne indurata, papules with surrounding tough, inflammatory infiltration (also with plasma cells and giant cells; see Ehrmann-Fick).

After the long continued use of iodine, bromine or chlorine, "excretory exanthemata" of varying kinds may appear, among them acne-like pustules with red margins. Tuberous tumor-like eruptions may also occur after the use of bromine, particularly on the thighs (Schaffer). The name ioderma (lit. by Fischel and Sobotka), or bromoderma, is used with the adjective tuberosum or fungoides (lit. in appendix). Chiari has described acne-like necrotizing skin inflammations in uremia.

A hordeolum is an acneform inflammation in a hair follicle of the eyelid.

Acne mentagra or sycosis, or folliculitis barbae, or barbers' itch, consists of papules and pustules from marked infiltration and suppuration of the hair follicles and their surroundings. The efflorescences are always pierced by a hair. Confluent and desiccated pustules may produce large scabs. The cause of sycosis vulgaris is the staphylococcus pyogenes (Ehrmann). In sycosis parasitaria the trichophyton fungus is found, see p. 2150.

Acne rosacea must be differentiated etiologically and anatomically from ordinary acne.

Acne rosacea appears in the face, particularly on the nose and its neighborhood (cheeks, forehead), especially in wine drinkers, but also from as yet unknown causes. The mildest form consists of reddening of the nose, forehead, the cheeks and the chin. In the beginning, the redness is evanescent; later it becomes permanent, and tortuous dilated blood vessels appear. From time to time, acne pustules are formed, sometimes only after the redness has persisted for years. In the more severe forms there are purple red, richly vascular spots, papules, nodules, rolls, par-

ticularly on the nose, but also sometimes in the cheeks. Microscopically, in addition to the changes in the sebaceous glands, which are quite similar to those of ordinary acne, there is dilatation of the blood vessel network in the various layers of the skin. Marked elephantic enlargement of the nose, particularly of the alae, is called acne hyperplastica, rhinophyma (fig. 1007). Microscopically, there is colossal enlargement of the sebaceous glands which is partly due to new formation (Kyrle); their excretory ducts are obstructed by secretion, and their neighborhoods are infiltrated by inflammatory products and fibrous tissue hyperplasias. Such cases give rather the impression of a soft elephantiasis with sebaceous gland hyperplasia, *i.e.*, a benign, new formation of sebaceous glands cystically dilated and enlarged, surrounded by richly vascular connective tissue (v. Bruns, lit. by Egger, R. Hoffmann, lit.), which may be removed by peeling off (decortication).

FIG. 1007.—Immense rhinophyma, man of 64 died from carcinoma of stomach. (Described in I. D., H. S. Egger, Basel, 1905, includ. microsc. picture.)

A **furuncle** is a circumscribed phlegmon of the skin running a typical course, and, as a rule, appearing in the neighborhood of the sebaceous glands; it is caused by the entrance of infectious organisms into a hair follicle (Unna), or, as many believe (Kochmann), into the sweat glands.

Furuncles do not occur in the palms of the hands and the soles of the feet. The so-called axillary furuncles, according to Fehrmann, are sweat gland abscesses, produced by a hidradenitis suppurativa; they do not have a pointed contour, but a rounded form; they are movable, never show plugs in the ducts, but subcutaneous fluctuations. Sweat gland abscesses may occur on the areola, around the anus, the labia majora, on the nape of the neck, the gluteal region, in general, wherever sweat glands are found (Schlatter).

Acne and impetigo staphylogenes may be differentiated from a furuncle by the degree of intensity of the inflammation, as well as in its spread to the subcutaneous tissue in which there are suppuration and partial necrosis of tissue; the causative organism is practically always the staphylococcus pyogenes aureus.

A furuncle begins as a tense, markedly reddened, tough, swelling, after which a vesicle appears on the surface containing a seropurulent fluid. This structure becomes pointed in the middle, and a purulent center appears around a white gray point. Then the pus breaks through, and the central part is separated and finally pushed out in the form of a

necrotic plug. This leaves a hole in the tissues, which is finally filled by granulation tissue as the suppuration disappears. There remains a punctiform, pigmented or pigmentless scar. Sites of preference: nape of the neck, nates, external genitalia.

Every furuncle, even the smallest, is uncertain as to its outcome (Riedel); there is always the danger of a thrombophlebitis (particularly in those of the face, chiefly the upper lip; p. 1809) and metastases by way of the blood stream. Constitutional diseases, such as diabetes, lower the resistance of the body to staphylococci (see also furunculosis after small pox, and after intestinal diseases, which are accompanied by abnormal fermentative processes, particularly in small children). (Therapy in those with a tendency to furuncles: polyvalent staphylococcus vaccine; preparations of yeast.)

A carbuncle is differentiated from a furuncle by its greater extent and its greater intensity. Anatomically, it is like a complexly built furuncle. A tendency to peripheral spread is characteristic of a carbuncle and helps differentiate it clinically from a furuncle. The course is longer, the separation of gangrenous tissue is much more important than in furuncles. Following the separation and removal of necrotic plugs, the surface of the skin may become pierced like a sieve, or else necrotic in toto; it is smeary, and soft, or undergoes desiccation and is lifted off by the pus underneath, whereupon the granulating wound surface is red and raw.

Usually the carbuncle remains confined to the cutis and subcutis, but a phlegmon of gangrenous character may be engrafted. Septicemia may cause death (Otten).

Etiology.—Acne pustules, furuncles, and carbuncles contain staphylococci as do most inflammations which have a tendency to localize; these conditions are also spoken of as staphylodermias (see impetigo, p. 2091); occasionally, however, streptococci, bacillus pyocyaneus, and proteus etc., are encountered. By tests with hemolysis, J. Koch believes he has shown that 90% of the staphylococci of the skin are harmless saprophytes, and that only three to five per cent are true pyogenic staphylococci. The production of hemolysin in a rabbit-blood-agar plate is said to parallel the degree of virulence of the organism; it is said to be higher in the case of staphylococci cultured from human abscesses, than in the ordinary skin staphylococci, (concerning the usefulness of hemolysis as a measure of virulence, see p. 1750). Furuncles occasionally occur in entirely healthy, usually younger, individuals. Repeated, numerous furuncles are seen in diabetics, in whom other skin diseases such as gangrene, are not rare. Garre experimentally produced furuncles in himself by rubbing staphylococcus aureus into the uninjured skin of the forearm. Thus, it can be assumed that the pyogenic cocci travel alongside of the hair into the

excretory ducts of the glands in the uninjured skin. This is well known to pathologists from autopsy observations, and agrees with the experience that such infections may be minimized by rubbing vaseline or similar substances into the backs of the hands and the region of the joints. Multiple torpid abscesses of the skin of sucklings may be caused by the entrance of staphylococci through the excretory ducts of the sweat glands (lit. by Lewandowsky). For the comparatively rare coccus metastases in the blood vessels of the skin, as for example, from an osteomyelitis, see Unna, Eug. Fraenkel, pp. 2072 and 2073.

The **pustula maligna,** or the anthrax pustule, is a local inflammation of the skin caused by the anthrax bacillus. Small injuries to the skin of the hand, forearm, the face, are the most frequent portals of entry; the bites of flies may also introduce the infection. The "carbuncle" develops in about fourteen days after infection as a small livid spot, which rapidly becomes red or yellow, as it proceeds to a hard boil of varying size, but perhaps as large as a hen's egg. On the top of the nodule there appears a small vesicle filled with water-clear or bloody serous fluid, the so-called anthrax pustule, from which an extensive serous or bloody serous infiltration may occasionally spread (anthrax edema). But a part of the skin may become a discolored scab which sinks below the surface so that its surroundings form a sort of wall around it; in the neighborhood, which is often little changed, but in some cases infiltrated, there appear water-clear yellow or blue red vesicles.

According to Treutlein, infection may actually occur through the hair follicles of the uninjured skin.

Anthrax infections are fairly frequent in the leather industry; the carbuncle is usually situated in the left neck, or the left nape of the neck (Felix, lit.; Rebentisch, in surgical therapy, "noli me tangere;" Pickenbach).

Microscopically, the papillary body and the corium are infiltrated down to and into the fatty tissue. In the upper parts, particularly in the papillary bodies, there is chiefly a fluid sero-fibrino-hemorrhagic exudate, which, as a rule, contains many anthrax bacilli, and which causes the formation of vesicles from solution and lifting off of the epithelium, whereas in the deeper layers, the infiltration is chiefly with polynuclear leucocytes, and bacilli cannot usually be found. The more richly cellular the serum, the more difficult it is to demonstrate the presence of bacteria.

Course of a Malignant Pustule.—If the process does not kill from blood infection (rare), it usually remains localized; the infiltrated tissue undergoes necrosis in greatest part, becomes demarkated, and granulation tissue and cicatrizing processes follow.

The rarer forms are: (*a*) the so-called external anthrax edema, and edematous inflammation without circumscribed pustule formation; black

scabs quickly appear on the edematous part (Bollinger). (*b*) Embolic skin anthrax proceeds with the formation of hemorrhagic spots, papules and vesicles, chiefly in the face and the trunk (Waldeyer, Weigert).

Occasionally a carbuncle leads to general infection with bacilli in the blood, whereby there is secondary involvement of the stomach or intestinal mucosa. General infection is much more frequent when the primary infection is in the intestinal tract (pp. 670 and 803), or lungs (p. 405). (Lit. concerning anthrax by Sobernheim, see also Martinotti.)

Necrosis and Gangrene of the Skin. (The expression "necrosis" and "gangrene" are used interchangeably. Thus, in general, we speak of carbolic acid necrosis or carbolic acid gangrene (Czerny), in spite of the fact that there is a dry gangrene or mummification in this condition.)

FIG. 1008.—Mumification of three toes of r. foot in arteriosclerosis; woman 47.

Simple necrosis, the death of circumscribed tissue parts, occurs most frequently as dry gangrene (mummification) in senility, particularly in the toes, and is produced by vessel changes; in mummification, the dead tissue imbibes blood coloring matter and becomes dry, the skin shrinks, becomes brown, blue, or black. Gangrene of the dry variety may occur in marasmus of various kinds, thus, after typhoid fever. In other cases, skin necrosis is embolic, *e.g.*, in heart failure. The entrance of organisms of decomposition produces softening; this is moist gangrene. Gangrene also follows freezing, third degree burns, the application of mineral acids or alkalies, the application of moist dressings even with weak (1–2%) carbolic acid, particularly on the fingers and toes, traumatic removal of skin; in diabetes the gangrene is caused chiefly by arteriosclerosis and consecutive thrombosis (Israel, Heidenhain, F. Koenig and others); it results from certain intoxications, *e.g.*, ergotism.

Syphilitic endarteritis may produce spontaneous gangrene, especially of the feet (Elsenberg, Haga), more often in young men or those of middle age.

A particular form is known as decubital gangrene or bed sores, which appear in the parts of the skin on which pressure has been exerted, most

frequently across the sacrum, the trochanters, the angles of the scapulae, but occasionally in other situations where the skin lies directly on bone or is compressed by tendons belonging to certain muscles. Decubitus depends upon capillary anemia and stasis in the compressed, or crushed, poorly nourished, tissues, which then become necrotic. Cardiac weakness, and certain spinal and cerebral diseases, particularly when sensation is diminished, favor the appearance of decubitus. The skin becomes blue black to black, dry and discolored, softened and smeary. The gangrene extends through the subcutis, the fascias, muscles, and bones. But this is not the only mode of formation; the necrotizing process may begin, conversely, in the deeper layers of the tissue and reach the skin the last of all (Wieting). According to Dietrich, portions of the gluteal muscles anemic from pressure are very important in sacral decubitus (Rossle).

Hospital gangrene is an inflammatory process with quick gangrenous tissue destruction which follows infection (anerobic bacilli, Matzenauer), of usually very small wounds (for instance, leech bites). The neighborhood of the wounds becomes dirty yellow, gray, gangrenous (Trendelenburg). According to Wieting, wound diphtheria (pp. 613 and 614), corresponds to the older conception of hospital gangrene.

> Gangrene-producing infections of the skin are apt to be associated with several organisms living in symbiosis, *e.g.*, the streptococcus and staphylococcus; see Brewer, G. E., and Meleney, F. L., Ann. Surg. 84: 438: 1926.

In **ergotism,** the gangrene is ushered in by blue-black discoloration and mummification, particularly of the fingers and toes with sequestration of the gangrenous parts. The vessels are filled with coagulated blood in the necrotic areas. There is a gangrenous form of ergotism as well as a convulsive form; both may occur coincidently; in the first, according to Kobert, the condition is due to the acid principle from ergot of rye, and the latter from cornutin, the alkaloid.

Perforating ulcer of the heel (Nelaton) usually begins in the soles of the feet near the balls of the toes, as a circumscribed thickening like an ordinary callous, often as hard as a nail. In the beginning, it is dead white; hemorrhage changes it dark to black; the surrounding tissue is not tender, but atrophic. There is then a sharply demarkated rounded loss of substance, which in many cases, shows a tendency to become a funnel-shaped ulcer with steep edges, extending occasionally even to the bones and joints. The condition is painless, stubborn, and readily recurs. The etiology is not uniform. It follows mechanical lesions, or obliterating vasculitis of isolated arteries of the feet, in which perhaps an ordinary

arteriosclerotic or syphilitic arteriosclerotic or alcoholic (?) cause is responsible (Lewai, Sternberg and others). In other cases, the ulcer appears in diseases of the spinal cord or peripheral nerves; thus, relatively frequently in tabes, syringomyelia, spina bifida, and even after injuries to the spinal cord or in general paralysis; then the term trophoneurotic inflammation, or neuroparalytic ulceration is used (Duplay, Morat, H. Fischer and others). But these so-called trophoneurotic influences (Cassirer, and p. 1070), are hypothetical in largest part, just as is the assumption of isolated trophic nerves and centers. Pressure, vasomotor and sensory irritants which may always be found as accompanying signs, as well as angiosclerosis, are no doubt the most important factors concerned. In leprosy, a perforating ulcer may appear. In certain cases, atrophy of the nerves has been observed; disturbances in sensation (anesthesia and analgesia) and trophic disturbances (epidermis hypertrophy and others) also occur in the neighborhood of the ulcer. The condition has occurred in children (lit. Tomasczewski), and in certain isolated cases an hereditary tendency has been observed (Gobell and Runge). Literature by Adrian; see also Levy.

Symmetrical gangrene may appear (*a*) as an independent disease (Raynaud's disease) as well as (*b*) in the course of other, particularly nervous, diseases; these latter are, among others, pathological processes in the spinal cord (Gobell and Runge, lit.), such as syringomyelia, diastematomyelia, tabes, tumors of the spinal cord or the nerve roots, epilepsy, hysteria (Towle, Rasch), and also exophthalmic goiter. Zambaco identifies the disease with leprosy, but Babes energetically protests this idea. As a basis for Raynaud's gangrene, there is believed to be an angioneurosis, and "pure" cases show no anatomic changes in the vessels and nerves (Harbitz). Semon lays stress, among other things, on the role of syphilis, since good results have been obtained in certain cases by antisyphilitic medication, but in contrast to the "pure cases," anatomical vessel changes are present, and the independence of the disease becomes quite doubtful. In those forms of gangrene discussed under (*b*), the above mentioned, so-called trophoneurotic influences occur. (*c*) During, or after infectious diseases such as typhoid fever, typhus fever, (Brauer), scarlet fever (lit. by Potpeschnigg), measles (lit. by Lutz), puerperal infection (Etienne and Wormser), pneumonia (Seidelmann), influenza (Soneck), diphtheria (Bailly, Thiele), syphilis, (see above), unilateral or symmetrical gangrene of the extremities, the fingers and toes is occasionally seen. As causes may be mentioned: embolism, autochthonous arteritic arterial thrombosis (see p. 120 and also Morgenstern and Gruber), venous thrombosis (lit. Barraud, Penkert, Todyo, Bull). Riedel described gangrene of the thigh following illuminating gas poisoning. Extensive literature by Gerlach concerning

juvenile gangrene. Concerning concussion necrosis of entire extremities with and without obstruction of the main vessels, see Kuttner and p. 2039.

Many authors reserve the name Raynaud's disease for independent gangrene. But this distinction cannot be made (Oppenheim), since the causes operating during the course of other nervous diseases such as tabes, syringomyelia, tumors of the spinal cord (Calmann), multiple sclerosis (Strauss) etc., may produce symmetrical gangrene. Three stages of Raynaud's disease may be differentiated; (1) syncope (regional ischemia), wherein a finger or perhaps the toes become waxy, pale and cold, (2) local asphyxia (regional cyanosis), the parts become blue to black, and (3) gangrene with final separation of parts, usually the distal phalanges of the fingers. Raynaud's disease, as a rule, is accompanied by violent pains which usually disappear after a certain number of months, and after the disease has passed through one of the above named stages, but they may disappear before the third stage is reached. Repeated attacks are not uncommon. More rarely, parts other than the fingers are attacked such as the point of the nose, the nates, the ears. (Extensive lit. and clinical application by Oppenheim. See Beck for case in a suckling; see also Kolisch, Osborne.)

Acrocyanosis chronica anesthetica (Cassirer), is a gradually developing asphyxia of the ends of the extremities which does not occur in attacks, and is thus differentiated from Raynaud's disease. It is believed to be related to the nervous system. (Lit. by Oppenheim.)

Acute, multiple skin gangrene, presumably because of a nervous influence (multiple neurotic gangrene), may appear suddenly without preceding local disturbances; the etiology is quite unknown. Hysterical manifestations are thought of, and also artefacts caused by deliberate burning of the skin (Ehrmann; see also Dinkler). Concerning true skin diphtheria, see under noma genitalium, p. 1693 and also p. 613 (lit. by Scheller, Reinhardt).

Artificial cutis necrosis, see Riecke.

Ainhum is a disease found chiefly in African negroes (Clark, da Silva Lima, Wucherer), which consists of the formation of a ring-shaped band around the fourth or fifth toe which becomes gangrenous, and falls off spontaneously, whereas the remainder of the body remains healthy. Zambaco hypothecates, of course without proof, that leprosy is concerned in the process.

Literature to the chapter on necrosis and gangrene is in the appendix.

Skin Ulcers.—There must be differentiated: (1) ulcers which are produced by the destruction of specific infectious granulation tumors, and thus produce true tumors of the skin, and (2) simple cutaneous ulcers.

The former will be discussed under their respective headings in later chapters. Simple ulcers (Kaposi, Riehl), are produced by loss of substance in pathological tissue; they produce a secretion. The surrounding part is composed of inflammatory, infiltrated, suppurating tissue and granulation tissue; the latter does not progress to cicatrization, but shows the same tendency to destruction as the former. Therein lies the difference between an ulcer, and a suppurating and granulating wound. The reasons for the progressive destruction in the ulcer, *i.e.*, for its diminished tendency to cicatrization, which is the end result of every ulcerative cutaneous defect, are external mechanical injuries (rubbings, pressure) or circulatory disturbances (varices, etc.), or unfavorable general conditions such as general anemia, marasmus, diabetes; or the ulcer itself remains infected permanently, is irritated anew, or contains within itself the tendency to destruction, as in tuberculosis, leprosy, syphilis, or a malignant new growth.

FIG. 1009.—Immense elephantiasis in leg ulcer, existing 16 years.

In every ulcer there may be differentiated: the base, the edges, the surroundings, the form, and the size. Frequent, important practical forms of cutaneous ulceration are:

Leg ulcers (see p. 169, fig. 95). Following stasis in the veins, and edematous infiltration and tension in the tissues, the skin becomes very sensitive, so that even the smallest superficial excoriations which may occur from very mild traumata, bring about first a superficial, then a deeper loss of substance and thus produce atonic ulcers. They produce torpid, pale red granulations, secreting smaller amounts of serous fluid, and as long as the fundamental disturbances remain, tend to resist healing over long lengths of time. If the ulcers do become cicatrized, the scars are thin and readily break open. In time, the ulcers may reach considerable extent, and lay bare the bones. On the edges, which ordinarily are flat and thin, enormous cicatrization, and bacony hardening (callous

ulcers) may develop, whereby the edges are made firm and tough. The surroundings are cyanotic, edematous, scaly, speckled, or diffusely pigmented, deep brown, and hardened with scars. Occasionally the foot and thigh become elephantic (fig. 1009).

See p. 1092 concerning the periostitis and ostitis ossificans so frequently a complication of leg ulcers, even to a superficial necrosis, or an osteomyelitis.

Atypical epithelial overgrowth is frequent in old ulcers, in the form of epithelial shreds between the granulations, without tendency to cover the surface completely or sufficiently. Not rarely, a carcinoma develops in the base of a florid leg ulcer, or in its scar. Corresponding to the extent of the ulcer, there appears a larger or smaller ulcerating squamous cell epithelial tumor (see the case fig. 96 on p. 169, in which the ulcer had existed for fifty years), which infiltrates into the tibia, and produces spontaneous fractures and metastases.

Ulcus molle, or soft chancre, is a contagious venereal ulcer (Sigmund), has nothing in common with syphilis, but may occur in combination with the primary lesion of the latter disease. It is a contagious local affection, acquired practically only by the sexual route, and therefore is most frequent in the genital regions, but may occasionally appear in certain other situations of the skin such as the legs, the hands, the arms, and the face. Twenty-four hours after infection from a chancroid secretion, there appears a red inflammatory area surrounding a cellular papule, which quickly becomes converted into a pustule. After removal of the covering of the pustule, a deep crateriform ulcer appears; its rounded or angular edges look as though bored out with a drill; its base is uneven and covered with mortifying tissue masses, but the base as well as the edges remain soft as long as the ulcer enlarges from progressive mortification. Much highly infectious pus is exuded which leads to autoinoculations, so that the almost constant occurrence of multiple "venereal ulcers" is explained. Later, the ulcer becomes clean, all dead tissue is removed, and it changes to a healthy, no longer infectious, wound.

The course usually lasts about six to seven weeks (Kaposi). As complications, there may appear erysipelas, wound diphtheria, and gangrene which produces the phagedenic ulcer. According to Matzenauer, an ulcer gangrenosum contains anerobic gram positive bacilli, which also produce a type of hospital gangrene. The non-complicated ulcers heal with the formation of flat scars. Sometimes the regional lymph nodes swell, and often rapidly develop a suppurative lymphadenitis (buboes). Bacteriology: a streptobacillus has been demonstrated by Unna in the secretions and tissues of soft ulcers. Probably this bacillus is identical with the Ducrey streptobacillus, which produces long chains of

rods lying partly between, and partly in the cells of soft chancres. This bacillus is said to be the specific cause of the ulcer (Pusey, Kruse, Buschke and others). Cultures on one part of agar, one part of rabbit's blood, and inoculation into animals and humans, have been successful (Lenglet, Besancon, Griffon and Le Sourd, Tomasczewski; see Rille, for the first culture on human blood agar). It may be stained with rongalit white (see Unna; Krantz). The buboes of soft chancres sometimes contain sterile non-infectious pus, but others contain virulent Ducrey bacilli, and typical soft chancres may be produced with the contents of the bubo. The "sterile" pus is bacteria-free according to Krefting, but Buschke states that it occasionally contains avirulent specific bacilli, and Adrian, that it practically always has this content. Clinically, the course of avirulent buboes is benign and rapid; the virulent infections, however, quickly change into melting buboes, and after lasting a week or two, become formed, ulcerating craters. The ordinary pyogenic organisms are not of importance in the production of a chancroid bubo (Tomasczewski, lit.).

Thalmann believes that some serpiginous, soft ulcers are probably caused by gonococci which may appear in the subcutaneous tissue as pus producers (Salomon).

The so-called **x-ray ulcer** appears after the entrance of leucocytes in the cell complexes of the skin, after they undergo degeneration under the influence of the rays. The epidermal cells degenerate most quickly and most seriously (Scholtz), in less degree the cells of the glandular organs, the muscles, the vessels, and the connective tissue. The chronicity of the ulcers is caused most probably by changes in the blood vessels, which Gassmann describes as a vacuolizing degeneration of the intima and muscularis, and which the author, who saw them in the preparations of Gassmann, believes are of considerable significance. Muhsam (lit.) saw an endarteritis obliterans. These ulcers are often preceded by a sort of erythema solare. The so-called roentgen carcinomas may appear from the ulcers produced by a roentgen-ray dermatitis (p. 2083), and also from the keratoses and simple atrophies, or more rarely from the intact skin itself, even years after contact with the rays (O. Hesse). They are often multiple and involve a number of fingers, both hands, particularly the dorsal sides, occur mostly in younger individuals, and show little tendency to glandular involvement and internal metastases (among others, see Allen, Wyss, Porter and White, lit., Porter, Rowntree, Coenen, F. Rosenbach, O. Hesse). It is said that sarcomas also arise in these situations (Pels-Leusden). Roentgen-ray carcinomas may appear in a lupus skin (see O. Hesse).

Granulations and Granulomas.— A granuloma is a tumor-like overgrowth, composed of a cellular and richly vascular granulation tissue.

Granulation tissue and its change into scar tissue (see pp. 9 and 10).

See von Gaza (lit.), concerning the biological importance of the granulation layer in wounds, which protects the body from loss of substance and from damage from the external world, particularly from bacteria. See O. Sachs (lit.), concerning granulation tissue and epithelial tissue stimulants of aniline nature, such as scarlet red, and many others.

Many granulomas develop without well defined causes such as quite mild injuries (traumatic granulomas); soft papillary or spongy overgrowths appear in the injured parts of the skin.

An interesting form of granuloma is seen occasionally in the new-born as a satiny, highly red granulation in the umbilical cord which may become as large as a pea; some of these umbilical granulomas are partly diverticulum prolapses (Kustner) which lie in relation to the ductus omphalomesentericus and contain glandular tubes, but others are true adenomas arising from rests of the ductus omphalomesentericus, (more details and lit. p. 726). [For diseases of the umbilicus in general, see Cullen, T. S. Embryology, Anatomy and Diseases of the Umbilicus with Diseases of the Urachus. Philadelphia, W. B. Saunders Co., 1916. Also, Cullen, T. S., Surg. Gyn. and Obst. 35: 257: 1922.—*Translator.*]

Certain other autochthonous umbilical ulcerations may be considered as sweat gland adenomas (W. v. Noorden).

The so-called telangiectatic granulomas (granuloma pediculatum), are not at all uncommon; they are blue red, almost always solitary, painless pedunculated, tumor-like granulomas, which vary between the size of the head of a pin and a small walnut, are elastic, tougher than simple granulation tissue nodules, and of rounded or mushroom shape; they have a short pedicle which inserts into the cutis, from which the epidermis travels upwards a short space somewhat like a collar. This granuloma seems to be thrust through the epidermis. The surface is smooth or finely granular, moist, or covered by a fibrino-purulent or crusty covering. Sites of preference: hands, particularly the palms, the ball of the thumb, the finger, the lips (p. 564), the angles of the mouth, the tongue (p. 564), soles of the feet, more rarely the shoulder or scapular region. According to the experience of the author, an injury usually precedes the growth (Martinotti), and a mild infection is practically always present. The telangiectatic granuloma begins in the red spot. Its peculiarity lies in its extraordinary richness of capillaries, which tend to become ectatic, so that it may be actually cavernous, a fact which may have to do with the narrowness of the collar of epidermis which surrounds it, and through which the pedicle, or the neck of the granuloma is inserted. If the capillaries are not quite so wide, their thick endothelial cells together with the spindle cells of the neighborhood resemble a sarcoma in its

cellular richness; Reitmann has, in fact, spoken of "angiosarcoma," but this is entirely erroneous. Nor can we agree with the idea that it is a peculiarly proliferating angioma (Konjetzny, Schurmeyer). When the ectasy of the vessels is present and there is a marked inflammatory cellular infiltration, the structure resembles ordinary granulation tissue. Clinically, the differential diagnosis between this and sarcoma is exceedingly difficult at times.

This granuloma pediculatum has often been called the human botryomycosis. Botryomycosis is a particular and specific disease, studied

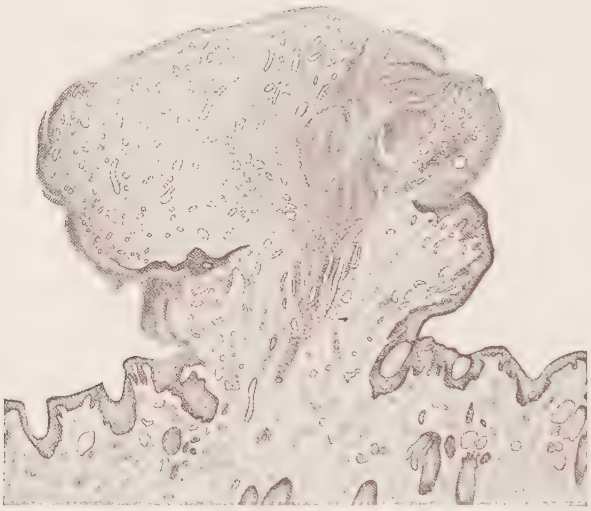


FIG. 1010.—Granuloma pediculatum (telangiectatic granuloma) of scalp of 64 year old man.

especially in horses in which the tissues of the infectious cutaneous granuloma contain micrococci in grape-like masses (Bollinger and Saul). While Delbet, Chevassu and Lenormant are quite reserved in speaking of this botryomycosis of humans, Letulle believes that its irritant is an ameba, which, after its death, is recognized as a rounded, hyalin mass with remnants of leucocytes lying around it; collections of these masses produce characteristic yellow granules; the suppurative form is said to be secondary to infection with staphylococci. Others, as for example, Kuttner, believe that staphylococci alone are important, and that collections of them produce the little globular excrescences or botryomyces; thus the condition is due entirely to an insidious infection with staphylococci. Schridde's case with cell inclusions which possibly contained, as he believed, the causative protozoa, was different, as Hoffmann and Konjetzny emphasized, from the ordinary telangiectatic granuloma, especially in the fact that the bones were involved. Heuck and others never found cellular inclu-

sions, but believe that there is infection, although they do not insist that the staphylococcus pyogenes aureus is the only causative irritant. See also Bennecke, Kreibich (lit.), Ferrarini, Herxheimer-Schmidt, Hadda. See Zurhelle, concerning granuloma pediculatum luposum.

Rocha-Lima calls attention to the similarity of telangiectatic granulomas to the infective nodules in the skin (and later, in various other organs) of verruga peruviana, a febrile disease occurring in Peru; in this case, the fundamental process is an overgrowth of the vascular elements.

Translator's Remarks.—Granuloma inguinale, first considered a tropical disease, but now known to be endemic also in temperate zones, usually begins as a small non-inflamed papule in the groin. This ruptures, exudes a small amount of cloudy fluid, refuses to heal and spreads slowly upward toward the crest of the ileum and downward to the perineum, sometimes to the buttocks, usually to the vulva, often to the penis, where it may be primary. It is an exuberant growth of soft granulation tissue, entirely unlike an eroded, undermining ulcer, but appears like an area of clean surgical granulation except for the exuberance. There is some tendency to cicatrization, but it spreads in other directions. It occurs predominantly in the negro, but cases in the white race have been reported. The diagnosis can be made by the fairly characteristic clinical picture of which chronicity and failure to respond to ordinary treatment are parts and smears which show numerous large mononuclear cells. Stained by Wright's or Giemsa's method, these cells show intracellular encapsulated bacilli originally described by Donovan and since called Donovan's bodies. These organisms have been identified by some as members of the bacillus mucous capsulatus group. Their causative relation is not accepted by all observers. The histopathology of the lesion consists of a superficial cellular area of young connective tissue, many endothelial leucocytes, a few polymorphonuclear leucocytes, many lymphocytes and an occasional eosinophile. New vessels are formed as in any granulation tissue, and the mononuclears as found in smears are present in considerable numbers. In older lesions, older connective tissue is found. The squamous epithelium is often hyperplastic around the edges and undermined for a short distance by the granulation. See Randall, A., Small, J. C., and Bells, W. P., Surg. Gyn. & Obst. 34:717; 1922.—McIntosh, J. A., J. A. M. A. 87:996:1926.

Granuloma fungoides (Auspitz), or mycosis fungoides (Alibert) may be differentiated into two types. In the classical form (1) after a vesicular

and desquamating eczematous pre-stage (so-called prefungoid symptoms or premycotic stage), which may last many years, there appear multiple, flat and nodular, rounded or crescent-shaped infiltrates in the cutis and subcutis; these swell, (a shorter fungoid stage), to dark red, mushroom-shaped, often broad based, or tomato-shaped, mildly lobulated and basket woven, tumor-like nodules from the size of walnuts to fists, with smooth or ulcerated surfaces and of soft elastic consistency, which often (just as a tomato) are composed of a more dense shell, and a half fluid content. Papillary tumors may also appear. Males, in their earlier

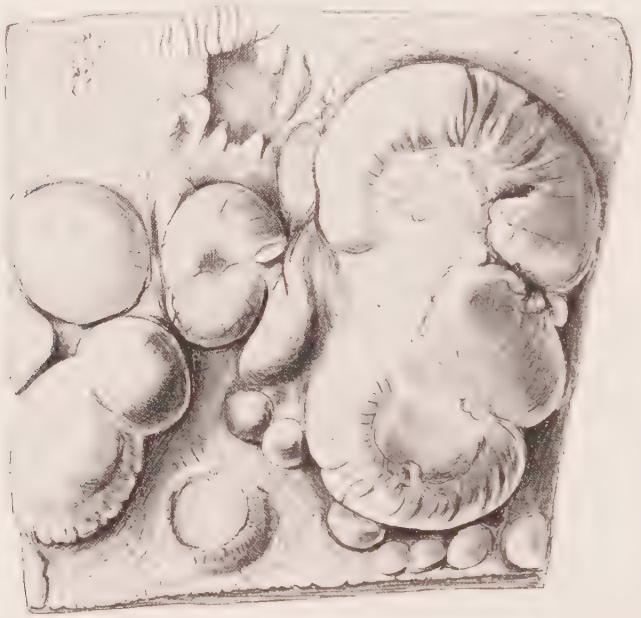


FIG. 1011.—Granuloma (mycosis fungoides) of skin of r. lower leg; some nodules are thick fungoid and tomato-shaped, some sunken in, umbilicated, others have undergone cicatricial change. From a woman 54 years old who had nodules in the face, one almost as large as a hen's egg in the eyebrow, one the size of a chestnut on the bridge of the nose, one the size of an almond at l. nasal angle, and two the size of a small cherry, one on lower lip and the other on the head, among the hair. Tuberculosis of genitals also present; nat. size.

years, are the most frequent victims. Mycosis fungoides d'emblee (2), has no eczematous pre-stage. The number of the nodules is usually less, and their boundaries sharper.

In severe cases, the nodules are everywhere in the skin; in others, the disease is localized perhaps at first in the face, the axillae, or on one leg. Pressure necrosis of the outer shell allows secondary invasion of bacteria, and softening or suppuration of the nodules; others, even those as large as small apples, regress, and disappear completely in an amazingly short length of time; this process usually begins in the central parts, while the peripheries are retained as wall-like circular, cresentic, or garland-like

structures; they may leave scars. This does not occur in sarcomas which always show progressive growth. Individuals who have reached the complete fungoid stage usually die in two to three years of marasmus or sepsis.

The question of "metastases:"—As a rule, the internal organs remain free. Paltauf first described a case in which there were foci in the pleura, the lungs, stomach, lymph nodes, showing the structure of granuloma fungoides, so that the impression of metastases was given. Paltauf and Zumbusch, and Paltauf and Scherber then described similar cases with foci in the various organs, thus in the heart, (p. 83), liver, spleen, kidneys, bone marrow, stomach, etc.; they did not consider them metastases, however, but simply parts of the symptoms of a general disease, which is usually localized in the skin. The external and internal lymph nodes are occasionally involved. While Unna considers that those cases which showed metastases were from a lymphosarcoma arising in a granuloma fungoides, Arzt, on the basis of two cases with metastases to the bones of large round cell sarcoma structure, pleads for the true malignant blastoma nature of the disease. Kuznitzky offers the hypothesis that perhaps the lungs, in which he demonstrated roentgenologic foci, are the portal of entry of an unknown infectious agent, and that the skin manifestations are metastases of these primary foci. Histologically, the new growth consists of fine fibrillar tissue—and therefore simulates lymphoid tissue—composed of polymorphous, round, and to a less degree, spindle-shaped cells; some round cells are like lymphocytes, others, perhaps predominant in number, are more rich in protoplasm, with large pale nuclei (like epithelial cells), and many mitoses; leucocytes are isolated; plasma cells and mast cells are often present in considerable numbers. Sternberg's cells are not found, in contradistinction to lymphogranulomatosis. Not only are the blood vessels retained in the granuloma but the capillaries are remarkably dilated, in contradistinction to those in tuberculosis and syphilis; in fact, they may form new vessels (details by Tryb). Unna lays particular stress on the prominent part played by the growing connective tissue of the subpapillary layer of the cutis, and thus on the true granuloma

FIG. 1012.—Granuloma (mycosis fungoides) of a young girl. Section through skin nodule. Prominent capillaries in granulation tissue. (Very low power.)

nature of the disease in contrast to mere infiltration. Leukemic tumors contain only small lymphocytes (Polland, see also Mariani, lit.). According to Tryb, local eosinophilia persists during the entire process, and usually more richly in the nonulcerated than in the ulcerated tumors; see Spiethoff for the eosinophilia in the blood; the blood picture is, however, not characteristic (Polland). The etiology is unknown; an infectious origin is hypothecated (lit. by Wolters, v. Zumbusch, Herxheimer and Hubner, Paltauf, Verroti, Sternberg). Those bacteria which have been found to date (streptococci in ulcerated skin tumors), were, no doubt, accidental invaders; in non-ulcerated nodules microorganisms were mixed (Lassar and Doenitz and others).

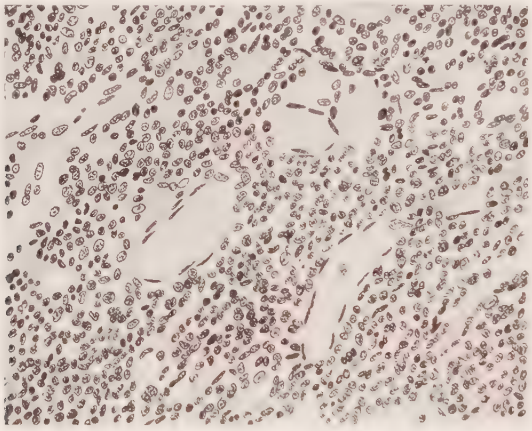


FIG. 1013.—Granuloma (mycosis fungoides); same case as fig. 1012, high power. Prominent wide capillaries.

Granuloma fungoides is more nearly similar to the (infectious) granulation tumors (see also Unna), and must be differentiated from (*a*) round and spindle cell sarcomas (but many speak of granuloma fungoides as sarcomatosis of the skin), (Kaposi); the failure of true metastases in the lymph nodes and internal organs is against this idea; the cells of a small cell sarcoma are also more uniform; Weber speaks of sarcoid, which, however, is an unlucky term. It must further be differentiated (*b*) from lymphosarcoma (p. 274), (*c*) from the aleukemic and leukemic lymphomas (p. 263). By many, however, it is said to be related to leukemia or pseudoleukemia; see opposed, Brandweiner; see also Sequeira and others concerning this point.

See under leukemia and pseudoleukemia in volume I, and the older literature by Linser, concerning lymphogranulomatosis cutis; literature on p. 269, concerning the leukemic and pseudoleukemic changes in the skin. See Knowle concerning the differential diagnosis from syphilis and tuberculosis. See p. 2125 concerning granuloma pernio, the so-called lupus pernio.

Translator's Remarks.—The translator's attention was drawn forcefully to the behaviour of liquid petrolatum when injected under the skin. A woman, 26 years old, complained of growing tumors on the external aspect of the left upper arm, stating that she had received several hypodermic injections of camphorated oil in this region during the course of typhoid fever ten years before. The presumption is that the camphor was contained in liquid

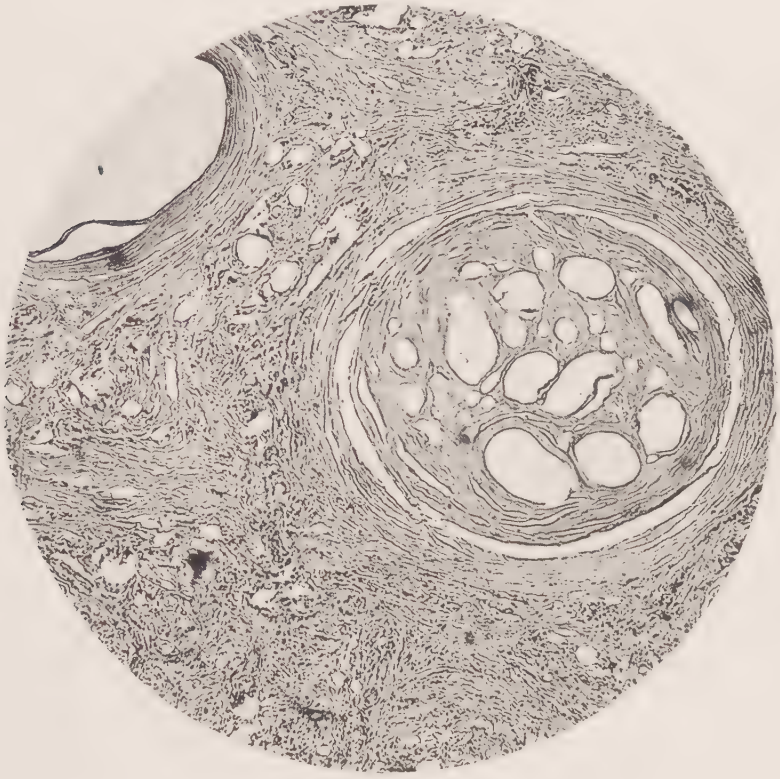


FIG. 1014.—Paraffinoma, see text. × 80. L. H. #19796.

petrolatum. Lumps at the site of injection never disappeared but continued to "grow" and produce new nodules. Excision of several proved them to be "paraffinomas" as shown in the illustration, (fig. 1014), *i.e.*, large spaces surrounded by granulomatous tissue with foreign body giant cells. The "growth" is probably of the encapsulating tissue; the appearance of new nodules may be due to a process somewhat as follows: larger globules of oil are broken up into droplets by the ingrowth of connective tissue, the latter then are encapsulated or broken up still further, leading to new nodules. Since, experimentally, the oil has been found in adjacent lymph nodes of laboratory animals, it is probable that

it is transported in humans as well. This transportation may be halted and new nodules form, etc. See Weidman, F., J. A. M. A., 80:1761:1923 and experiments by Weidman, F. D. and Jefferies. M. S., Arch. Dermat. & Syph., 7:209:1923.

Infectious Granulation Tumors

Tuberculosis of the Skin. —Various forms may be described: **lupus,** or lupus vulgaris, or tuberculosis luposa, is the most frequent form of skin

FIG. 1015.—Lupus vulgaris. On right hand of Lettish girl 18 years old. Present for three years. The granulations were proved tuberculous by histologic study. Practically completely healed after about three months. Local treatment with ultra-violet light and general treatment with cod liver oil and other tonics. There were enlarged lymph nodes at the elbow and in the axillae, slightly tender, which likewise disappeared when the local lesion healed. L. H.

tuberculosis; it is a chronic disease, characterized by the primary efflorescence of a lupus papule, which is a conglomeration of miliary tubercles lying in an inflammatory infiltrate. The process is much more frequent in the female sex, begins mostly in childhood, and always runs a very slow course. In later years, it becomes rarer, possibly because a certain

amount of immunity has been built up, due to the fact that most individuals have a certain amount of latent or active internal tuberculosis (Lewandowsky). It appears most frequently in the face (nose, cheeks, upper lip, lids, muscles of the ear), but it may be seen in other, usually uncovered, situations, such as the extremities. Neighboring mucous membranes may be invaded by a primary skin lupus. But primary mucous membrane lupus, particularly in the nose, is not uncommon, and the skin may be secondarily involved from this.

Lupus may also arise from a tuberculous focus under the skin which later heals, thus, *e.g.*, from tuberculous tendovaginitis, lymphadenitis, orchitis, epididymitis; in lupus of the region of the elbow, adherence to the healed bone points to a relationship with bone disease (Lang).

Fig. 1016.—Lupus vulgaris; a, epidermis proliferating downward here and there. Especially to the right papillae cut across; b, cutis with lupus new formation, in the cellular tissue are nodules of epithelioid cells containing giant cells (c); d, perivascular cell strands; e, ulcer. (Low power.)

This form of tuberculosis described as lupus may be confined to the skin, or it may be combined with tuberculosis in other organs, particularly the lungs, in the conditions grouped under the name of scrofula (p. 260, lit. by Forchhammer).

Histologically, lupus is characterized by the appearance of typical miliary tubercles containing inflammatory infiltrated tissue; the cells of the latter are polynuclear leucocytes and plasma cells (fig. 1016). The nodules become differentiated in the richly cellular tissue, inasmuch as isolated cell masses appear instead of rounded epithelioid cell forms; nucleated giant cells are occasionally found in great numbers. The tubercles themselves usually contain but few stainable tubercle bacilli (Hidaka, lit.), but always Much's rods and granules (Boas and Ditlevsen). The cell foci may become homogeneous or finely granular masses, *i.e.*, undergo

caseation. The tuberculous granulation tissue begins in the neighborhood of blood vessels, and often spreads in the form of bands along the larger cutaneous vessels; the spread takes place into subcutis as well as into the papillae. The elastic fibers disappear where it is established, and calcified, iron-containing remnants of the latter may be preserved inside of giant cells. The epithelial structures of the skin, particularly the rete Malpighii, are, at first, uninvolved; later the epidermis becomes infiltrated and destroyed; sometimes, however, the epidermis grows, but not particularly; the rete drives pegs into the cutis and the atypical epithelial overgrowths resemble carcinoma. (See also Miyahara.)

W. Busch describes this as an epithelioma-like form of lupus; in spite of this, however, its course is no more malignant than that of ordinary lupus. But secondary carcinoma may appear in the edges or in the base of a florid lupus (lupus carcinoma); the author, for example, examined such a preparation from the lower eye-lid, and another from the cubital region of a seventeen year old girl. Clinically, the onset of carcinoma is manifested by a change of the typical, slowly progressive disintegration to a rapid, local, intensely malignant destruction; metastases are rare, even in the regional lymph nodes, cachexia appears quite late. Carcinoma may appear even in the scars of lupus. (Statistical by Ashihara, Sequeira; see also Jadassohn, Zweig, Silberstein, lit.) According to K. A. Heiberg, atypical epithelial growths are said to have larger nuclei than those of normal tissue, carcinoma still larger, even over fifteen micro-millimeters.

In a certain stage the nodules are not prominent, but may, at times, be felt through the skin; infiltrates are formed, which produce soft, dark blue or brown red, smooth, or rough spots in the skin from the size of heads of pins to lentils (lupus maculosus). If caseous destruction with resorption takes place in these lupus spots, they shrink and leave flat, cicatricial depressions with a torn, desquamating epidermis (lupus exfoliatus resolutivus). In lupus tuberosus nodosus s. tumidus, the nodules are prominent in the cutis from the size of millet seeds to peas or larger. More rarely, this form disappears leaving a cicatrix (lupus fibrosus). Still more marked prominences, composed of nodules of particularly luxuriant diffuse granulation or connective tissue overgrowth in the cutis and subcutis, bring forth the so-called lupus nodosus hypertrophicus. Occasionally, not rounded, but papillary, warty proliferations, partly under the epithelium and partly in the base of the ulcer, are caused by overgrowth of the papillary body; they are composed of epithelial overgrowths; the surface is covered with scabs and epithelial flakes (lupus papillosus s. verrucosus); this is also spoken of as the epithelioma-like type (Th. Naegeli); this form prefers the hands and feet.

Occasionally lupus appears like an elephantiasis arabum; occasionally in the face, as a lupus follicularis (Fox), whereby acneform efflorescences with a brown gelatinous appearance without inflammatory changes are found (Finger).

In the rarer, tumor producing, lupus there are sharply outlined soft, livid red, new growths, even as large as a small apple (lit. by Heuck).

Lupus pernio, particularly that of the face, more rarely of the hands or the ears, produces sharply outlined, tumor-like foci, somewhat similar to frost bites because of their venous hyperemia, and marked vascularization in the periphery and centers. The older views stated that lupus pernio was a sub-form of ordinary lupus vulgaris, which developed a close relationship to the vessels and to venous stasis and vessel dilatation (Neisser-Jadassohn). Kreibich, and others, such as Ehrmann, Polland, Bloch differentiate the disease from lupus vulgaris because of the criteria of lupus pernio: failure to find tubercle bacilli, lack of central necrosis, the finding of merely isolated giant cells, very sharp demarcation of the nodules, which become actually encapsuled by connective tissue, failure of reactive infiltration around them, as well as final intactness of the epidermis; the foci consist of practically nothing but epithelioid cells with a few plasma cells. Zieler (lit.) differentiates (*a*) pure cases, *i.e.*, chronic infectious granulomas which have nothing to do with tuberculosis, *i.e.*, granuloma pernio, (see also Bruhns and Alexander) and (*b*) combinations with tuberculosis. Grouven clings to the older viewpoint. Literature by Zieler, Hilgers, who recommend the terminology "erythema pernio" as suggested by Zieler; Lewandowsky, Grocz.

Superficial nodules frequently rupture through the skin and there are added suppuration, necrosis, and the formation of ulcers, which are covered with pus scabs; new nodules appear in the edges (lupus exulcerans, vorax, exedens). The ulcers may spread in their depths or in their diameters. Ulcerative destruction of lupus infiltrates is practically always the rule.

If the ulcers heal, there remains a deforming, wavy scar. Loss of substance, especially in the face, is quite frequent. The point of the nose is particularly involved. In extensive lupus, the entire face may be covered with scars, and the nose, lips and eyelids are destroyed in largest part. It is characteristic of lupus that new infiltrations appear in the scars. Spontaneous healing (resolution) is quite rare. In many cases seemingly healed, the injection of tuberculin may demonstrate reacting lupus tissue. See p. 2084 concerning the results of the treatment of lupus with Finsen's light. Usually the process progressively involves new areas, and the entire cartilaginous structure of the nose is destroyed

as the disease progresses during the entire life of the patient. If new peripheral nodules are formed around a lupus scar as a center, the condition is known as lupus serpiginosus. Lupus, mostly papillomatous, on the extremities, can, by extensive marked sclerosing scar formation, lead to elephantiasis from plugging of the lymph channels (Gaillard and Bouffle, Daniel, lit.). Lupus may spread from the skin to the tendon sheaths, periosteum, the joints, and produce mutilations, *e.g.*, of the phalanges, usually the distal; but crippling is also produced by the contraction of scars, interference with growth, changes in neighboring joints and bones (subluxation, luxation, contracture), (Guterbock, Kuttner).

According to Audry, lupus of the skin of the extremities is secondary in the majority of cases to tuberculosis in deeper parts such as the bones and joints.

The so-called **scrofuloderma** (tuberculosis subcutanea colliquativa), occurs particularly in the faces, necks, backs of the neck of scrofulous children, but also in older individuals in certain predisposed parts of the skin; it is the most frequent form of skin tuberculosis. In the subcutis and the deeper layers, localized nodular foci and strands of tuberculous granulation tissue are formed which contain but few tubercle bacilli, and giant cells, and which secondarily spread to the remaining skin. The skin is movable at first, but as the subcutaneous nodule extends into it, becomes more and more adherent, thin, and blue red. The tuberculous tissue undergoes quick and extensive necrosis, probably due to an intensive activity of the toxin of the bacilli. By colliquation of the caseous masses, the foci become soft like abscesses; the thin skin is widely undermined, often ruptured in many places; a thin yellow caseous pulp is emptied with many destroyed leucocytes but no pyogenic organisms. Thus there appear fistulous ulcers with livid edges and necrotic bases, which run a very torpid course, and heal with markedly deformed ridges and pointed scars as in lues.

Origin.—Infection of the subcutis may occur by extension from neighboring lymph nodes, or tuberculous fistulae from the joints, bones, the anus, etc. Unna described all secondary tuberculous conditions of the skin as scrofuloderma, even tuberculous skin fistulae, which permanently convey tuberculous tissue debris from an internal focus; he calls them a cylindrically growing scrofuloderma. According to Arzt and Kumer (lit.), hematogenous origin is probably fairly common.

In another form of skin tuberculosis, viz., that described by Riehl, there is infiltration into the cutis, with fungoid overgrowth or ulceration but the formation of nodules is absent, (tuberculosis fungosa cutis). Doutrelepont described a form in which solitary or multiple tumor-like nodules appear.

(Concerning lichen scrofulosorum, see p. 2101; concerning the so-called lupus erythematosus, see p. 2099.)

Tuberculosis miliaris ulcerosa is characterized by the appearance of miliary nodules, which soon undergo caseation and ulceration. The ulcer is shallow, with a pale red or a yellow base, in which miliary nodules are occasionally seen; the edges are finely gnawed, and in general but little infiltrated. The ulcer enlarges by serpiginous progression and rapid destruction of the (lymphoid cellular) tubercle, which does not have time to become macroscopically sharply identified as a nodule. The edges are usually but little infiltrated. The number of tubercle bacilli is very large, as a rule. Localization: usually in phthisical individuals, the ulcers prefer the neighborhood of the bodily openings, are practically always accompanied by advanced tuberculosis of internal organs, and appear obviously from autoinoculation of bacteria-containing secretions and excretions of the phthisical individuals.

If, for example, as on the penis, the ulcer becomes chronic, with hardened edges and base, it may simulate carcinoma. Baumgarten described a disseminated skin tuberculosis in the form of small papilloma-like outgrowths. In general miliary tuberculosis of the skin, Heller, Leichtenstern, Hedinger, and others (lit. by Jadassohn), saw fine red points and rounded, pointed papules, some of which underwent regression and others which were transformed into vesicles and blebs, filled with a cloudy yellow content and tubercle bacilli; they are believed to be hematogenous miliary tuberculosis of the papillary bodies. The same is true of lupus follicularis disseminatus faciei (lit. by Arndt, Lowenberg). Such observations show that a hematogenous genesis of ulcerative tuberculosis of the skin is possible (see histological details by Leiner and Spieler). See Brugsgaard concerning acute disseminated skin tuberculosis in childhood.

Local inoculation lupus, the best known form of which is the post mortem tubercle, is peculiar not only in regard to its origin, *i.e.*, it is inoculated, but also in other respects; it is somewhat similar to lupus verrucosus (verruca necrogenica tuberculosa or tuberculum anatomicum); they are seen particularly in anatomists, pathologists, and other individuals who become infected from phthisical cadavers or from tuberculous animal material. The bacilli are inoculated into small cutaneous wounds. The post mortem tubercle appears as a pustule or an ulceration, in a slowly growing, blue nodule which later is covered with a superficial scab, or by papillary, warty tissue. They are found practically only on the hands, particularly the backs of the hands, and the forearms. They are the most benign form of lupus, and occasionally heal spontaneously. [See Stokes J. H., Am. J. Med Sci. 169: 722: 1925.—*Translator.*]

Microscopically, there are overgrowth of the papillary bodies and a thickening and atypical growth of the epidermis as an expression of the tuberculous inflammatory process. Very rarely, the subcutis is involved. In addition to low virulence tubercle bacilli, (Karg and others), pyogenic cocci are often found; these may lead to suppuration of the tubercle and to the formation of pustules, or, occasionally, to lymphangitis. A lymph node tuberculosis is rare. A particular form of inoculation lupus which occurs in those who handle tuberculous cattle, has been described by Riehl and Paltauf as tuberculosis verrucosa cutis, a papillary tuberculous focal disease, which is usually more benign than ordinary lupus, but in spite of this, as Neisser remarks, the detection of bacilli in the foci composed of granulation tissue with giant cells, is usually much easier than in ordinary lupus verrucosus. Fabry observed them on the backs of the hands of workers in coal mines and believes the condition is not true tuberculosis, but a tuberculid. Among others, the author saw an exquisite case of this kind in the form of a stubborn tumor as large as a pigeon's egg, on the sole of the foot of a fifty-two year old man. Jordan reports lymph vessel tuberculosis as a relatively frequent complication. H. Fuchs describes luetic foci which could not be told clinically from tuberculosis verrucosa. Among other forms of inoculation tuberculosis, there may be mentioned that arising from puncture of the ear lobes, from the ritual circumcision, the rubbing in of saliva during tattooing, the washing of tuberculous clothes, injuries from broken sputum cups used by consumptives, the use of the hyperdermic needle, after having previously been used in phthisical individuals (Bruns); in general, vaccination tuberculosis of the skin appears in the form of a cadaver tubercle or as an ulcer. General infection is ordinarily rare, and usually occurs only in those hereditarily handicapped; but in the tuberculosis following circumcision it is said to be more frequent (see under penis).

Concerning tuberculids, see the remarks on p. 2101 under lichen scrofulosorum (lupus erythematodes), also under erythema induratum on p. 2079. Zieler (lit.) and others believe that the tuberculids are actual bacillary tuberculoses, like the well recognized skin tuberculoses; he, however, claims that the tuberculids represent hypersusceptibility reactions of the skin in tuberculous humans from metastasized tubercle bacilli, which, as a rule, lead to the destruction of the bacilli. Barthelemy, who believes in a primary disease of the follicles, remarks that folliclis is one of the benign tuberculous skin infections, or tuberculids; it is charac terized by the repeated appearance of tough nodules in the depths of the skin, the size of millet seeds to beans, which disappear after leaving a brown discoloration, or which ulcerate and heal with the formation of small white scars. A papulo-necrotic tuberculid is also spoken of. Sites of preference

are the ears, particularly the edges of the pinna, the elbows, the knees, the region of the calves, the back of the foot. Alexander believes that folliclis as well as erythema induratum are small deposits of circulating, mildly virulent, tubercle bacilli, and he postulates that a tuberculous focus can always be found in the body. The positive results of inoculation of guinea pigs, as performed by Leiner and Spieler, give evidence of bacillary origin. Literature by Alexander, Zieler; see also Leiner and Spieler, Jadassohn, Lewandowsky.

Boeck's "sarcoid" consisting of nodular foci, which on pressure under glass become yellow brown and are traversed by telangiectasias, show microscopically large cell epithelioid and giant cell nodules sharply outlined; it is probably a particular reaction of the organism against the virus of the tubercle bacillus. Similar nodules may appear in the lymph nodes and in the spleen (Lewandowsky, lit.). The belief of Kuznitzky-Bittorf, that it is a non-tuberculous disease sui generis, but one of the so-called pseudoleukemic general diseases, has aroused violent opposition from Frieboes, among others (lit.).

It must be remembered that foreign body tubercles (p. 870) produce cellular nodules around all sorts of foreign bodies, such as threads from operations, hair, etc.; the nodules are composed of epithelioid and usually numerous giant cells which are often dominant. In contrast to tubercles they do not show caseation. Mistakes may, however, be made when the foreign bodies are no longer present.

Syphilis.—We will discuss the following: (*a*) the initial manifestation of syphilis, *i.e.*, the primary effect produced at the situation of infection and (*b*) the syphilids, *i.e.*, actual cutaneous syphilis, or the dermatoses brought about as a result of the general infection.

a. **The Primary Syphilitic Effect.**—The initial manifestation of syphilis may be genital, paragenital, or extragenital on any situation of the body surface, *e.g.*, a finger, the areola, the scalp, (very rare, lit. by Herrmann), the alae of the nose, the eye-lids, in, or around the mouth, even on the tonsils. It is usually solitary, rarely multiple.

When multiple, the lesions are not contemporary but more often successive developments. Thus, in a case of Sack, there appeared fifteen primary effects on the penis and scrotum, caused, no doubt, by auto-inoculation favored by scabies and balanoposthitis.

The primary lesion occurs in various forms, the most important of which are (1) the more frequent initial sclerosis, (2) the rarer initial papule.

In the initial sclerosis, there may be differentiated: simple, eroded, ulcerated, and gangrenous sclerosis, and similarly, the initial papule may be differentiated into simple, eroded, or ulcerated forms. This makes about seven types; added to which, according to Lang, there may be

recognized (8) weakly developed infiltration, (9) seemingly bare erosion, and (10) rhagades. But even with these, all forms have not been described; there is, *e.g.*, a combination with non-syphilitic diseases such as simple venereal ulcers which is called the mixed chancre, or with variola vaccinia (vaccination-syphilis).

The initial sclerosis (*a*) develops after an incubation period of about three weeks as an infiltration with mild reddening, sharp boundaries,

FIG. 1017.—Syphilitic primary lesion of prepuce removed fr. patient; hemispherical flattened. In the corium, especially in the papillae, perivascular, but also interstitial leucocytic and plasma cell infiltration and connective tissue proliferation. The walls of blood vessels are filled with cells. The non-infiltrated fibrilla bundles are hyalin (sclerotic); isolated vessel walls are also hyalin. The epidermis is partly filled with leucocytes and plasma cells (beginning erosion). (Moderate power.)

which to palpation are as hard as cartilage, and as though a small plate had been imbedded in the skin; it causes little pain. Usually, the sclerosis is flat, more rarely dome shaped, spherical, cylindrical or spindle shaped.

Histologically, there are found (fig. 1017): a thick infiltration, sharply demarcated from the normal surroundings; in point of time, at first with leucocytes, then with lymphocytes, and later with plasma cells (p. 9); this, in contrast to the polymorphonuclear infiltration and the more diffuse boundaries in soft chancres, is of differential value. After a few

weeks there is always an overgrowth of the fixed connective tissue cells of the cutis. At first, the process is perivascular, later it is spread between the vessels and the papillary bodies, and all through the spaces of the cutis and subcutis; the above mentioned white blood cells and spindle cells appear with epithelioid cells and perhaps even isolated giant cells between them. The smallest blood vessels, particularly the veins, show cellular infiltration of their neighborhoods (perivasculitis), and overgrowth of all their coats, particularly the intima (endovasculitis), threatening acute and complete obliteration. But the blood vessels in the very immediate vicinity of the sclerosis are entirely intact. The still visible bundles of fibrillae of the cutis appear hyalin (fig. 1017); this helps to cause the hardness of the initial sclerosis (Zurhelle, however, believes that the hardness is helped by a marked development of the reticulum fibers). The lymph vessels are likewise surrounded by infiltrates; newly formed capillaries appear among the latter; not rarely their lumena are closed by rounded, and epithelioid cells (Rieder, Nobl); the process is therefore a peri- and endo-lymphangitis. The epidermis invades between the papillae from the infiltrate in irregular prolongations and shreds. The boundaries of the papillary bodies may be obscure and disappear; cells from the corium then infiltrate into the epithelial layers (fig. 1017), which become more and more thinned, as layer after layer is lost until they finally disappear entirely (erosion). The lesion is then presented as a red, lacquered, shallow defect which, after treatment, even when the infiltration is still present, is readily covered by new skin.

The etiological relationship of the spirochaete pallidum (Schaudinn-Hoffmann 1905), is now admitted by the great majority of workers. The spirochaete pallidum (p. 1286, fig. 667), is the cause of syphilis; it may be found two weeks after infection in streaks and sectioned preparations, in the deeper layers of the bordering epidermis, as well as in large numbers along the course of the blood and lymph vessels of a large percentage of primary lesions; in the central parts of older primary lesions it cannot be found, because it is destroyed directly, or by phagocytosis (details by Pirila). (Their significance has always been disputed by Saling, who believes they are shrunken nerve ending fibrillae; see W. Schulze, Siegel and others.) The reality of the spirochetes and the fact that they move in three dimensions can easily be demonstrated by the dark field illuminator; furthermore, spirochetes can be demonstrated by the India ink method, whereby one drop of India ink thinned with distilled water (1–9), is mixed with a drop of the suspicious tissue fluid, then spread upon a slide, allowed to dry, and examined with the oil immersion lens. The spirochetes are easily demonstrated as white streaks upon a black background, but it is well to be careful not to diagnose the spirochetae

pallida merely upon morphological grounds. See Schereschewsky, Muhlens, Sowade (lit.), concerning cultural experiments; for pure cultures and experimental inoculation of rabbits, dogs, monkeys, see Noguchi, Hoffmann, Schereschewsky and others.

The older Levaditi method of demonstrating them in sections is as follows: very small pieces of tissue are fixed in 10% formalin, then placed in 95% alcohol for twenty-four hours, then in distilled water until the pieces sink, after which they are allowed to remain three days in 1½% to 3% silver nitrate solution in the incubator. A short washing in water is followed by forty-two hours in a mixture of pyrogallic acid, 4 grams, formalin 40%, 5 cubic centimeters, aq. destill. 100 cubic centimeters. Wash in water. Sections may be made by the frozen method or paraffin, or celloidin imbedding. The spirochetes are black brown to intense black, impregnated with silver. Counter staining is not necessary (illustration p. 1286). See Hage for Fontana's silver method; for other methods (carbol fuchsin staining), see Becker.

An ulceration appears upon the sclerosis when the infiltrated cutis and epidermis undergo destruction. This form, the ordinary Hunterian chancre, is very frequent, and is produced by secondary pyogenic cocci. It appears as a shallow, shell-like ulcer with a hard base, partly or entirely surrounded by a hard, sharply bordered edge. The base of the ulcer is either discolored, and covered with remnants of tissue, or granulating and bright red.

A small amount of slimy pus is secreted by the ulcer. More rarely, granulation tissue grows from the base, fills up the ulcer and grows over into the neighborhood (ulcus elevatum). There are also cases in which the initial infiltrates become changed into gummatous masses, and thus to a serpiginous ulcer.

In initial papules the cellular infiltration and the cellular new growth are located chiefly in the papillary bodies. The nodule tends to force itself upward instead of against the cutis and subcutis. Small initial papules as large as peas, are often hemispherical, red and livid; larger ones are flat or plateau-like and even. The initial papules are less hard than the scleroses.

They may be differentiated from the sclerosis because the lymph vessels remain intact in the papule. The spread of the infiltrate is dependent upon the branching of the larger arteries; the larger branches of the veins are involved in the form of a peri- and endophlebitis.

Healing of the Primary Lesion.– After suitable medication, the sclerosis slowly heals during the course of weeks or months, but even for years, a connective tissue thickening marks the original site. Occasionally a cicatricial, atrophic, pigmented situation remains. Papules are

practically completely absorbed, and only rarely are they lastingly organized.

b. **Cutaneous Syphilis.**—Syphilids (syphilitic dermatoses), appear as symptoms of congenital, as well as acquired syphilis. Their recognition is all the more important, since they are the first positive symptoms of a generalized infection.

General Observations Concerning the Forms Taken by Syphilids.—According to the classical description of Kaposi, the various forms taken by the syphilids, whether spots, papules, pustules, ulcers and crusts, may be exactly similar to many non-syphilitic dermatoses, so that neither the polymorphy of the efflorescence, nor its localization on the flexor surfaces, nor its itching or failure to itch, can always be used as differential points. There are, however, three characteristics which are general to all the various kinds of syphilids: first, there appear sharply outlined, thick, even, cellular infiltrates, and new formation of connective tissue cells in the corium and the papillary bodies. The sharp boundaries appear, because there is no surrounding inflammation to produce a gradual transition into the neighborhood. Secondly, the cells are frail and do not have the ability to produce a lasting organization, but show a tendency to regression and to disappearance, either by fatty degeneration and absorption, or by softening into a purulent mass. Thirdly, the direction and course of events through which the cellular infiltrates and the regressive changes pass are always constant; the infiltrates enlarge in the centrifugal direction; the disappearance begins centrally and proceeds toward the periphery.

Thus the basic type of a syphilitic nodule is the papule. This appears as a sharply bounded, thick, cellular infiltration in the papillary body and corium. The infiltration extends somewhat into the neighborhood, is glistening, since the epidermal covering is under tension, is tough, and brown red, as a result of the exit of blood from obstructed vessels. Then the regressive changes begin; at first the central part sinks; the epidermis becomes wrinkled and disappears in scales. The periphery remains tough, under tension, glistening, and sharply bordered from the healthy skin. If this process occurs in papules situated quite close together, but distinct as individuals, (each papule with a central desquamation), which later become somewhat confluent, there appears the picture of psoriasis diffusa (plantaris and palmaris). In roseola syphilitica, these three criteria must be modified to a certain extent. The infiltration is negligible. The roseola are the pre-stages of the papules, with which they are often combined. If the central destruction occurs with superficial suppuration, scabs are formed by a drying of the secretion along with epithelial debris. The suppurative destruction may lead to pustules on the tops of the papules. According to whether vesicles or bullae are formed, the condi-

tion is called herpes or pemphigus syphiliticus. Even in syphilitic cutaneous ulcers, with their characteristic appearance, these three cardinal peculiarities may be recognized. The ulcer appears as a central loss of substance in a nodule, *i.e.*, an infiltrate. The bases and edges are still surrounded by a hard infiltration, as though covered with bacon. The edges are tough, sharply defined and perhaps somewhat undermined. This ulcer embedded, so to speak, in the cell masses, becomes covered with a scab; if this is lifted off by an accumulation of fluid, the progressive destruction is covered by a new scab larger than the old one; this is repeated, until the picture is that of a rupia syphilitica; a centrally raised scab becomes surrounded by a larger ring of scabs (Kaposi). But peripheral infiltration is present, and after lifting off the scab, the characteristic ulceration becomes visible. If the zone of infiltration is not completely around the circumference, but present only to about two-thirds, the free one-third may show, as a healing process, the growth of granulation tissue from the neighborhood after the specific infiltration is destroyed. Meanwhile destruction continues on the other sides, and the kidney-shaped form of ulceration appears, which, if continued, gives rise to the picture of serpiginous syphilitic ulceration.

We will now rapidly sketch the chief forms of cutaneous syphilids:

(*a*) **Roseola syphilitica** consists of red, not sharply marginated, spots, moderately prominent, some of which disappear on pressure, of the size of the heads of pins to finger nails; they do not itch. Points of preference are the trunk and the flexor surfaces of the extremities. They are rare on the backs of the hands and feet. They remain days, weeks, without further changes in form, and disappear without desquamation in from three to four weeks, usually leaving no trace; more rarely, they last for months and leave brown spots. Roseola is hard to see on a dark skin (black brown to deep black). When the infiltrations are more marked, they produce the frequent maculopapular syphilids of the early stages.

Roseola is usually the first manifestation of constitutional syphilis; it appears six to twelve weeks following infection, also as a recurrent symptom inside of the first year, in the forms of roseola annularis, serpiginosa, gyrata, etc. An important point of localization, according to Lang, is the inner surface of the preputial sac and the glans. Roseola occasionally produces a syphilitic discharge (balanitis), which may easily be confused with an ordinary balanoposthitis, especially if the roseola has disappeared from the remainder of the body, or if it is a recurrent syphilitic erythema which involves only the glans. But alongside of the diffuse redness produced by the decomposed sebum, there can still be found the sharply bordered, circumscribed, flat syphilitic infiltrates which become eroded, and from which most of the secretion comes. In situations

covered by hair (head, eyebrows, axilla, pubes), the roseola may lead to defluvium capillorum. Usually, it takes the form of small, bare spots, more rarely large, bare spots. Occasionally, changes in the internal organs with diseases of the mucous membranes, iritis, nephritis, orchitis, even splenic tumor, may appear coincidently with a roseola.

The maculae disappear, perhaps leaving behind smooth pigmentless spots (leukoderma syphiliticum). Leukoderma (p. 2165) is of some practical importance since the pigmentless spots which may be retained for months and years, represent previous papules.

(*b*) **Papular Syphilids.** Two forms may be distinguished: the large papular or lenticular, and the small papular or miliary. The lenticular

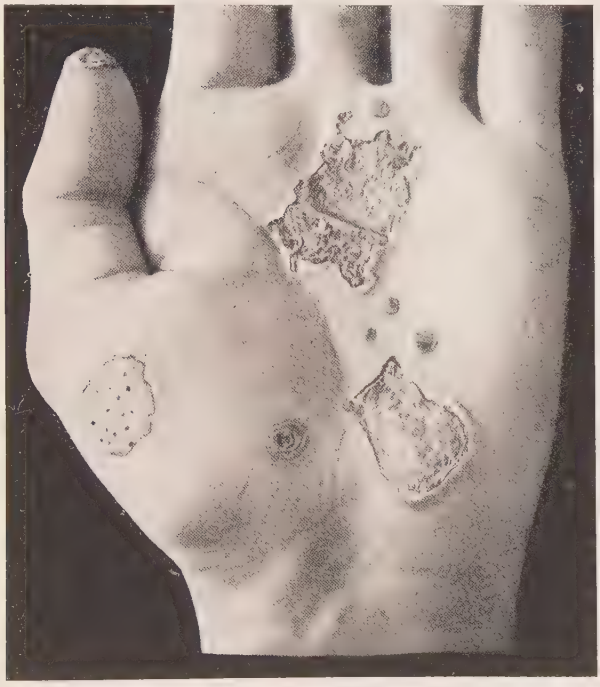


FIG. 1018.—Palmar syphilitic psoriasis; wax model; 4/5 nat. size.

form combined with roseola is often the first symptom of constitutional syphilis. The papules may also develop from the roseola. They are sharply bordered, brown red, (ham red), tough, glistening and somewhat prominent. On dry areas the papules remain dry and desquamate moderately; in moist areas they become moist. If the involution of the syphilid is delayed for a long while in the stage of desquamation, the condition is called papulo-squamous syphilid.

Pustules or crusts (papulo-pustular or papulo-crustous syphilid), also are formed occasionally. The papular syphilid is the most frequent

form of recurrence during the first five to ten years. In the beginning it is more universal, in later years, more local. Occasionally single giant papules are developed in severe cases. Their points of location by preference are: the trunk, the face, particularly the chin and the naso-labial fold and also in the angles of the mouth, the scalp, particularly at the edges of the hair (corona venerea), in the genitals, the perineum, anus, the axillae, the flexor surfaces of the larger joints. In the palms of the hands and the soles of the feet, papules produce small circumscribed red areas and quite flat infiltrations, over which a markedly cornified epithelial layer is stretched and slowly undergoes desquamation. Usually both palms or both plantar surfaces, or both palms and soles together are involved. Clavus-like hyperkeratotic situations are particularly frequent on the heels. (Psoriasis palmaris et plantaris or syphilis papulosa palmae et plantae; for differential diagnosis from psoriasis vulgaris, see p. 2098.) Recurrences of this syphilid are quite common but usually not symmetrical; it may, however, be the first sign of constitutional syphilis. Ulcerated papules appear in the folds of the toes. Psoriasis of long duration leaves dirty brown discolorations.

Fig. 1019.—Plantar syphilitic psoriasis; wax model.

Condylomata lata s. papulae latae s. plaques muqueuses; weeping, moist papules (papules madidantes) develop in situations which remain moist from pathological or physiological secretions, thus, in the folds of the skin, as in the external genitalia, the anus, the axillae, the folds of the breast, around the areola in lactating women, the angles of the mouth, the anal region of sucklings, etc. The papules are of the size of pennies to dollars, like slabs or plateaus, perhaps confluent; at first they are more or less reddened, covered with gray detritus when they secrete a viscid fluid

which usually contains many spirochetes; later they become bluish and dry. They may also ulcerate. These are among the most frequently observed of the recurrent syphilids, particularly during the first two years, although they may appear later, even after ten to thirty years and in cases well treated (Fournier). They are highly contagious; from auto-inoculation they spread to the surrounding skin. Occasionally, they produce a primary lesion similar to that of the initial sclerosis (p. 2130), inasmuch as they are contagious. This is of practical importance, since, if present in sucklings and in the breasts of lactating women, the question may be asked if they are recurrences of an older syphilis, or a primary lesion appearing from three to six weeks after infection (Kaposi). The regression of these papules takes weeks to months.

The small papular syphilid, the so-called lichen syphiliticus, is seen particularly in poorly nourished individuals (scrofulous). They appear as small, brown red, almost miliary, papules arranged in heaps, or groups, or in streaks. They last longer than the lenticular forms. Involution follows with marked desquamation; healing occurs with small scars in the form of shallow punctiform depressions.

(*c*) **Pustular syphilids** develop by softening of the papules of a papular syphilid, usually after the entrance of staphylococci. It occurs particularly in poorly nourished individuals. There may be differentiated: a large pustular and a small pustular syphilid. In the first, the pustules are as large as beans; papules crowned by a pustule (syphilis papulo-pustulosa), and papules without pustules may appear side by side; other papules show a central scab (syphilis papulo-crustosa).

Pustular syphilids occur: (1) in syphilis maligna, occasionally in the early period, as the first symptom, accompanied by high fever; in this type a maculo-pustular secondary stage is generally entirely lacking and various sized ulcerations appear in the skin and mucous membranes, usually within the first year. Fever, anemia, headaches, periosteal swellings, are accompanying symptoms. There are cases, (as the author has seen at autopsy), which resist every therapy; the demonstration of spirochetes in the typical foci is usually unsuccessful, but vaccination into apes is possible (Buschke). Pustular syphilids occur (2) as a recurrent eruption of early syphilis; these eruptions may have a universal distribution, and (3) be late recurrent pustular eruptions which, on the other hand, are usually localized (nose, forehead, capillitium). The course, as a rule, is serious in contrast to the ordinary pustules. After removal of the covering or the scab, the resulting ulcer has a mildly infiltrated base and edges. Healing occurs with the formation of a deep smooth, more rarely a ridged, scar. Occasionally the base of the ulcer shows flat, or plump papillomas, and produces gland-like, glistening red,

juniper berry-like, or encrustated, or epithelialized overgrowths, which may become larger than a silver dollar, and which ordinarily undergo regression (framboesia syphilitica).

Tropical framboesia must be differentiated from syphilis; the former is a disease named from the polypapillomatosis of the skin; it does not confine itself to this condition, but is manifested in its later stages, just as syphilis, by gummata and ulcerations of the skin, bones, joints, mucous membranes, particularly as a rhinopharyngitis mutilans. In the papillomas of the skin, Castellani found the spirocheta pertenius (treponema pertenus); see Mayer. The differentiation from syphilis, with which it has nothing to do in spite of its external similarity (Schilling), and in spite of the fact that it is quite responsive to treatment with salvarsan, is not always easy; according to Hallenberger (lit. and illustrations), the point on which particular stress must be laid, is the histological demonstration of the lack of syphilitic vessel changes in the products of framboesia. See literature by Plehn.

Pemphigus syphiliticus is distinguished by particularly large pustules. A form similar to varicella or variola levis, is called varicella syphilitica (umbilicated pustules); one similar to acne, inasmuch as it is arranged around the hair follicles, is called acne syphilitica (slow course, ulcerations, healing with deep scars). According to Lang, acne syphilitica is difficult to distinguish from acne necrotica (Boeck). In a case of the latter seen by the author, the acne was particularly well marked on the trunk and the legs; papular prominences were hardly present, but the scabs of dry necroses were situated on mildly reddened situations as large as the head of a pin on a level with the skin. Gradually developing pustules as large as dollars, produced from flat infiltrates, lead to rupia (p. 2134). This usually occurs in advanced stages of syphilis. Often only one rupia pustule is present. After healing, white scars are formed, or merely atrophic scar-like defects in the skin. The complications in the rest of the body, which, in addition to maculae and papules, may show a pustular syphilid, are likewise disposed to suppuration (iritis, periostitis, ostitis, etc.).

(*d*) **Gummatous syphilis,** *i.e.*, true syphilitic gummata, are differentiated by their being larger, but not different fundamentally from papules, inasmuch as a peculiar granulation tissue with vessels is formed, partly able to change to scar tissue. See illustration and microscopic details, p. 2047.

There may be differentiated: a superficial and a deep lying gumma, or (1) a syphilis cutanea gummosa, multiple infiltrations and nodules in groups the size of beans and larger and (2) subcutaneous gummata. Both forms show transitions. At first, subcutaneous gummas are movable

under the skin, but later invade the cutis. In the beginning, the gummata are tough and elastic; later they are softer, and tender to pressure.

(1) **The cutaneous gummata** tend to run a very slow course, and appear as flat, hard, infiltrates similar to the initial sclerosis. In favorable cases they regress, leaving a scar-like thinning, and a sharply bordered, white glistening, depression of the skin. Very often, however, an ulcer develops —the ulcus gummosum—with a dirty base, sharp sloping, sometimes undermined edges, as though a drill had been at work. These are usually very painful. Older ulcers heal as new foci appear, often with an outspoken tendency to arrangement of the nodules into ovals, rings, arches, windings, kidney or sickle-shaped forms. The shallow depressed scars of the conglomerate cutaneous syphilids, are separated by bridges of retained skin corresponding to their early origin from distinct gummas; this is a very characteristic picture (fig. 1020). One large retracting scar results from a softened gumma. The scars resulting from deeply ulcerated syphilids, with marked tendency to granulation, become thick and furrowed.

Fig. 1020.—Tumor-like cicatricial gumma (clinically mistaken for sarcoma); at the back of the elephantic r. lower leg, covered with serpiginous ulcers, numerous ulcerated holes at the height of the prominence correspond to gummas several centimetres wide. On the other lower leg were deep brown serpiginous pigmented scars. Woman of 58 years with dry caries of roof of skull and gummatous pachymeningitis; pulmonary tuberculosis.

Scar-like defects remaining after the absorption of superficial syphilitic infiltrates (especially gummata), maculae atrophicae, are caused chiefly by a general atrophy of the skin tissues.

(2) The **subcutaneous gummata,** on the other hand, in contrast to the flat superficial, produce voluminous nodules up to the size of a fist, over which the skin remains unchanged and movable; later the gumma becomes fixed to the cutis or to the underlying tissues. Usually they are isolated nodules, particularly on the forehead, the nape of the neck, the back and the thighs. Sooner or later the center of the gumma undergoes softening, after which the process extends to the periphery; it then takes on a pseudofluctuating consistency. On incision a sticky, viscid, gum-like mass is emptied. Resorption of the gumma may take place after fatty degeneration; if this occurs in the center, this part falls in. Gum-

matous remains may undergo caseation and calcification, and become encapsulated by connective tissue. Ulceration is very frequent. The edges of a deep ulcer are blue red, thickened, sharply bordered and undermined, while its base is covered and infiltrated with necrotic shreds. Less commonly, cicatrizing gummatous overgrowths take on a tumor-like appearance, and may lead to mistaken diagnoses for malignant tumors (see figs. 1020 and 1022).

After healing, the edges become flattened, the base undergoes granulation, and produces pure pus. The result is always a scar, usually wavy, with pigmentation in its neighborhood. See microscopic illustrations of a subcutaneous gumma on p. 2047.

The course of gummatous syphilids is usually slow; exceptionally, it is rapid; in either case they lead to destruction, particularly of the nose, lips, scalp, or hands. As a further stage, necrosis of neighboring portions of cartilage or bones, mutilations, and even erosions of larger arteries are seen.

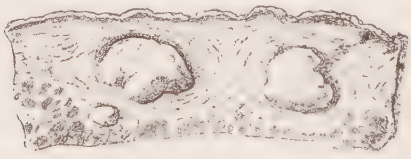


FIG. 1021.—Subcutaneous gummas of lower leg from upper perforated area of specimen, fig. 1022; nat. size.

FIG. 1022.—Severe gummatous ulcerative syphilis with radiating scars; lower leg of woman 26 years old; began 4 years before with a rounded nodule as large as a thumb; clinical diag: cancroid; amputation: from the same case as fig. 1021.

Skin gummata appear usually in the later stages of lues, in fact, are characteristic of late syphilis. Occasionally, however, they occur even in conjunction with secondary syphilids. Gummatous products are often simultaneously present in the osseous system, nervous system, mouth, pharynx, nose, larynx, etc. Lupus deposits may occur in infiltrates which persist for a long time, and do not undergo complete resorption. More rarely the gummatous ulcer becomes carcinomatous. Carcinoma may also appear side by side with the gummata, particularly in the tongue (v. Langenbeck). Even where a primary lesion has been present, there may develop a carcinoma; see under tongue, pp. 556 and 570.

Many dermatologists differentiate tuberous syphilids (grouped syphilids, papules or nodular syphilids, tertiary papules), from true gummas. Both of these forms of tertiary cutaneous syphilids, are often masked (Lang, Kaposi and others), as a subcutaneous, true skin gumma or as a superficial cutaneous gumma. But it must still be proved that the grouped syphilids can be anatomically divided as sharply from gummata as Unna and Tommasoli assume. Clinical subdivision may be justified by the classification: (1) tuberous syphilids, those in groups, often of serpiginous form, in some cases with ulcerative destruction (tubero-ulcerous syphilid), which gradually reach neighboring parts of the skin, with a flat spread, central healing, progressing into the periphery; and (2) actual gummata, of solitary distribution, with ulceration by perforation of the skin, tendency to the formation of spherical forms, and no tendency to central healing.

In the differential diagnosis, sporotrichosis (de Beurmann), and hemisporosis (Gougerot and Caravin) must be kept in mind, as discussed on p. 1147, and later on p. 2153; these diseases produce changes easily mistaken for gummatous as well as for tuberculous processes, particularly because the fungi are very difficult to demonstrate in the tissues.

The trypanids, *i.e.*, cutaneous manifestations in the white race of sleeping sickness (trypanosomiasis), produce papular or plaque-like or ulcerative lesions, quite similar to those of syphilis (Thiroux and p. 2158); oriental boils (Leishmaniosis ulcerosa cutis, see p. 2158), as well as blastomycosis cutis, may at times be mistaken for syphilis (p. 2154, and the case of Finger).

The **cutaneous syphilids in congenital or connatal** (incorrectly called hereditary) **syphilis.** (The term hereditary, inherited, should only be used when a condition is carried in copulation by the spermatazoa or ova. Such a germinative transmission has not been shown for syphilis. Transmission to the embryo occurs rather through the placenta. Good summary of this question by P. Schneider.) Because of their intense practical interest, these changes produced by the transmission of lues from the parents to the child in utero (from placental transfer), must be particularly described. (*a*) Early symptoms: appear at birth, or during the first three weeks of life, as a maculo-papular syphilid, not differing in its essentials from that of acquired syphilis, but proceeding with rhagades at the angles of the mouth, the anus, and the interdigital folds (p. 2136). Pemphigus syphiliticus is rarer; larger pus vesicles appear on ulcerated flat papules. According to Kaposi, a peculiar and characteristic form is a diffuse infiltration of the soles of the feet, and the palms of the hands, the skin of which becomes evenly brown red, dry, glistening, with rhagades here and there. (*b*) In later years, the same gum-

matous infiltrates and ulcerative syphilids appear in congenital as in acquired syphilis.

See Kralta (exhaustive lit.), concerning the fate of congenitally syphilitic children high mortality; see also Husten; see A. H. Hubner for congenital lues and psychic and nervous diseases such as idiocy, chorea, juvenile tabes, dementia praecox and other psychoses, which occur in about 26% of cases.

As pathognomonic of congenital syphilis there may be considered further: eye diseases,, such as keratitis parenchymatosa, chorioiditis areolaris (atrophic foci in the chorioidea and pigment overgrowths); particular deformities of the teeth known as Hutchinson's teeth: hypoplasia, which leads to half-moon shaped central excavations of the cutting edges of both upper and middle incisor teeth, accompanied by an arresting of the teeth in their lateral and longitudinal growth as a result of which the teeth become basket-shaped or even screw-shaped.

False Hutchinson's teeth are those which show merely a half moon-shaped excavation but no changes in the crowns; they may appear from hypoplasia, half-moon shaped caries, also from ordinary wearing down (Bogrow).

The question of the pathognomonic significance of Hutchinson's teeth is much debated. Enamel hypoplasia alone is not positively indicative of syphilis (see p. 584). The accent must be laid upon the simultaneous arrest of growth in the entire tooth, (lit. by Hochsinger, Moro, Noegger-ath, Sabouraud; see also Davidsohn, Kranz, Kraupa).

Fine linear scars, according to others, pseudo scars, radiate around the mouth and the eyes, and extend to the cheeks, the nasolabial folds, and the chin. Lang considers the scars the result of previous destructive processes; Silex believes the skin is pulled in by muscle tugs, and remains stationary because of the changes produced by the lues; Poor believes they are true scars, the consequence of removed infiltrates. The old Hutchinson triad, the above mentioned tooth anomalies, parenchymatous keratitis, and labyrinth deafness, are not entirely positive of congenital syphilis, but they do occur in this condition (Lang). Certain changes in the nose may be quite characteristic; the entire nose is too small, or has a saddle-shape, or has fallen in, or been pushed in at its root; see p. 298. Other signs, such as persistence of the childhood habitus, saber-shaped tibia, etc., are unreliable. See also the bones in congenital syphilis, p. 1133, lungs, p. 462, liver, p. 937.

Leprosy.—Leprosy, lepra arabum, or elephantiasis graecorum, is a chronic specific infectious disease, which produces yellow red to dark brown spots, and tough, flat, diffuse infiltrates or nodules in the skin and the bordering mucous membranes. The face and extremities as well

as the nerves are preferred. Hyperesthesias and anesthesias appear, and all sorts of diseases of the internal organs; with rare exceptions, leprosy leads directly or indirectly to death from a specific marasmus.

The lepra bacillus (Armauer Hansen, 1874, 1800), is the actual cause of the disease, and human beings, as most authors assume, are its only hosts. From the time of Damsch's vaccination into the anterior chamber of the eye of rabbits, there have been recorded many, more or less doubtful, experiments on the transmission of leprosy to animals (lit. by Fambri). In more recent works, Sugai, Nakano and others report successful transmission to mammals; others describe the cultivation of the leprosy bacillus, and the transmission to animals by means of a pure culture (Duval; see also Bertarelli). But in all these cases merely localized nodules were formed, while general symptoms were always absent. Reenstierna (lit.) claims he brought on a generalized leprosy as early as from forty-two to fifty-nine days. Kyrle produced only local lepromas in apes, but believes that he excluded a mere foreign body reaction of the animal to the vaccinated material, and that the leproma appeared because of actual increase in the number of the bacilli. The question of the natural occurring transmission from human to human is still open. Many believe that direct infection is possible through bacillus-containing secretions, others that the chief portals of entry are the nasal and mouth cavities, which may be the chief source of the infection; see Merian and Solano, and p. 300 for doubts of this idea (Boeck). In 96% of married couples in which the one was leprous, the other, even after years of intimate contact, did not become leprous. Furthermore, it is questionable if an indirect, intermediate transmission is possible, or that the bacteria which are excreted in the sputum, sweat, or feces (Boeck) may retain virulence, and after reaching the exterior, remain alive in the ground or on food (Arning). It has even been suggested that the virus must undergo a certain amount of development outside of the human body in order to produce leprosy, and that cattle (Engelbrecht) or insects (Hofmann, Lindsay, Sanders, Blaschko) may be its intermediate hosts. But all of this is very uncertain; most of the problems of leprosy still remain unanswered.

Inheritability must still be demonstrated. Leprosy was considered entirely incurable (see International Leprosy Conference, Berlin, 1897 and International Dermatological Congress, Berlin, 1904); but more hopeful news is at hand (International Leprosy Conference, Bergen 1909). Isolation of lepers in colonies, is a procedure ratified by the tests of many hundreds of years as a curb to the disease. (Lit. concerning leprosy by Sticker.)

While the combination of leprosy and tuberculosis is not rare (Fambri, lit.), leprosy and carcinoma are said practically never to occur together (Lewin, Soegaard).

Two chief forms may be differentiated: (*a*) nodular leprosy (cutanea), and (*b*) anesthetic leprosy (nervorum), also called trophoneurotic or nervous leprosy; these forms may be combined. According to Kaposi, a spotted leprosy may be added as a third form. Other subdivisions may be made and will be discussed below.

(*a*) *Nodular leprosy*, leprosy tuberosa, is found chiefly in the face—forehead, nose, lips—as well as in the terminal, particularly dorsal, parts of the extremities, also in the scrotum and prepuce.

According to Hansen and Looft, the scalp, the glans penis, the palms of the hands and the soles of the feet are immune; Rille (lit.), however, describes a case of leprosy tuberosa localized in the soles of the feet. (Similar to lues!)

It usually begins first in the eyebrows (the hair falls out), by the formation of spots (lepra maculosa), which represent cellular, infiltrates of the cutis and subcutis extraordinarily rich in bacilli. Following hyperemia, the spots are red, brown, sepia, or bronze colored, and glisten as though covered with oil. As time progresses, the infiltrates undergo some regression, and leave white, light gray, or brown, flat, irregular spots (morphaea alba and nigra) and the skin appears checkered. It may, however, become sunken and cicatricized. The spots may disappear in the centers, and progress peripherally. In its further course, this form may become the tuberous as well as the anesthetic. Extraordinarily slowly, in months, or even in years, these infiltrates become prominent in various situations of the body as nodules. When the latter are situated close together, they may coalesce to nodular plaques or trabeculae several centimeters thick.

The face, with its rolls on the forehead, prominent lips, nodular thickenings in the nose, cheeks and chin, and its thick jelly-like transparent nodules in the lobes of the ear, takes on the appearance of facies leontina. Spread to the conjunctiva and cornea is frequent and results in blindness. The mucous membranes of the nose, mouth, pharynx, larynx, often show changes (see under the special organs). Among the internal organs, there may be involved the liver, the spleen, more rarely the testes, the serous membranes, the bone marrow, probably by way of the blood stream, as demonstrated in certain cases of leprous bacillemia by de Beurmann and others, and by Rabinowitsch, who discovered lepra bacilli in the circulating blood six times in eight cases; other organs, as a rule, are not involved (Hansen), but, in certain cases, lepromas were found in the intestines, the lungs, the kidneys, exceptionally in the pancreas, the adrenals, and in the ovary (lit.); in short, they have been discovered in practically all the internal organs (leprosy universalis). Death has occurred from sepsis.

In many cases the nodules undergo spontaneous diminution in size, or disappear completely with the formation of scars wherein fatty changes are important, but, in contrast to tuberculosis, caseation never occurs. Not rarely, external conditions produce ulcers (leprous ulcerosa) characterized by an exceedingly lingering course; after remaining in statuo quo for years, they gradually disappear. They are often covered with skin, only to break down anew. Suppurative inflammation complicates the disease, particularly in the extremities, and leads to necrosis and demarkation with subsequent removal of isolated bone parts, or even entire members (lepra mutilans).

The leprous new growth is very frequent in the nerves, and spreads centripetally in the epi- and perineurium (Thoma); this produces anesthesia, often of larger areas. Further spread occurs by way of the lymph stream, and the submaxillary or inguinal nodes may be changed to enormous nodules showing a tendency to fatty changes.

W. and R. Haberfeld describe generalized granulomas of the lymph nodes in latent leprosy (nasal mucous membrane).

Microscopic Findings.—In the beginning, the lepromas are composed of granulation tissue which passes through the corium as a network of bands and appears from the neighborhood of the blood vessels and their walls. These cellular strands are most numerous where the greatest number of blood vessels is found, as in the superficial layers of the skin, around the hair follicles, and cutaneous glands. Later they coalesce, produce the diffuse leprous cell infiltrates of the cutis, and invade into the subcutaneous fatty tissue, wherein enormous infiltrates are produced. Among the cells of a leprous granulation tissue, round cells such as lymphocytes and plasma cells and fibroblasts are found at first; later, the epithelioid fibroblasts develop into the so-called leprous cells (Virchow), which are large, rounded, or oval with vacuolated, rounded inclusions, or are completely hyalin. Their vacuoles contain lipoids and occasionally cholesterin, thus producing an appearance similar to that in xanthoma cells (Cederkreutz). Leprous cells predominate; they may coalesce and produce large, lumpy, so-called lepra nodules (Neisser)—yellow masses which, according to Cederkreutz, are practically structureless and contain degenerated cells and nuclei, vacuoles, and many lepra bacilli; this "gloea" (Hansen) is met in the central part of a leproma. In addition to this, there may be present isolated giant cells of the Langhan's type (Schaffer), lit.). These lepra cells are the chief site of the bacilli (Hansen, Neisser, Cornil and others), which may be present in enormous numbers in cigar-shaped masses, or closely approximated strands, so that the cells appear completely filled. But bacilli also lie between the cells. In comparison to tuberculosis and syphilis, the bacteria-containing, vacuolated lepra

cells are quite characteristic (Thoma). According to Bergengrun and Kanthak, they are not actual cells but merely bacillary thrombi in lymph vessels which irritate the endothelial cells to overgrowth and enlargement into protoplasmic masses; bacilli occasionally lie within the cells but only accidentally. But this idea has brought forth nothing but criticism. Lepra bacilli, surrounded by a fatty capsule, are quite similar to tubercle bacilli and stain similarly (p. 422), but more lightly and more quickly; they are also decolorized more easily by acid, and as a means of further differentiation, can be well stained by simple watery dye solutions. These methods

FIG. 1023.—Lepra nodosa; skin of neck. Leprous granulation tissue has penetrated from the cutis broad and deep into the subcutaneous fatty tissue; man of 55 years. (Pub. by Storch, V.A. 148, 1897). (Quite low power.)

of differentiation are not, however, very reliable. Arning and Lewandowsky advise the use of prolonged Gram staining, as recommended by Much for tubercle bacilli; by this method it has been possible to demonstrate the lepra bacilli in the anesthetic form, a feat formerly impossible. By the silver impregnation method of Bertarelli, Yamamoto found the lepra bacilli unstained, whereas tubercle bacilli were stained black (Fambri).

(*b*) **Lepra Maculo-anesthetica.**—The anesthesia appears in the region of nodules and spots, or with other symptoms of leprosy, or, more characteristic, without other visible external changes of the skin, or with pemphigus-like blebs (pemphigus leprosus), which, after healing, leave glistening anesthetic situations in the skin, or after removal of their covering, shallow or deep ulcers (Kaposi). Skin and muscles undergo atrophy in situations

totally anesthetic. Injuries readily occur and lead to ulceration. By this means, or through the disappearance of the tissue, there are produced mutilations of various kinds. An entire foot or an entire hand may fall off quite suddenly (for more details, see p. 1147).

As the cause of the anesthesia, there is direct involvement of the peripheral nerve fibers in some of the cases by the spots and nodules in the cutis. In other cases, there is an independent lepra nervorum, which leads to atrophy of isolated fibers, and to paresthesias, hyperesthesias and anesthesias, also to consecutive trophic disturbances of the skin which becomes flat and glistening, hairless, without glands, etc., and further to falling off or splintering of the nails, (for bones p. 1147, joints, p. 1263). (Hansen and Looft, lit.)

See pp. 1981 and 2111 for the idea that Morvan's symptom-complex and Raynaud's symmetrical gangrene are related to leprosy, and also the idea of Zambaco who believes that all extraordinary or uncommon multilations—ainhum, for instance—are forms of leprosy. (Lit. concerning leprosy by A. v. Bergmann, Hansen and Babes.) [For the leprosy problem in the United States, see Denney O. E., Pub. Health Reports, reprint 1080.—*Translator.*]

Rhinoscleroma is discussed in more detail under nose (p. 300); there are established in the skin of the nose and bordering parts, hard, gray red nodules covered by epidermis, with later development of similar pictures in the nasopharyngeal or the laryngo-tracheal mucous membranes.

Glanders.—Infection through an injured place in the skin (nasal or cutaneous glanders of horses), produces a local process which leads to phlegmons, suppuration, gangrene, lymphangitis, lymphadenitis, pustules and ulcers, or a general infection (p. 299); the latter runs a septicopyemic course (lit. by Arzt, see also Herzog); embolic glanders foci appear by hematogenous routes, particularly in the lungs and in the spleen and kidneys; these more nearly resemble abscesses than nodules; other foci are innumerable muscle abscesses and cutaneous pustules, from the size of millet seeds to peas (may be mistaken for small pox, smears show glanders bacilli!), furuncles, hemorrhagic nodules, abscesses of varying sizes. (May be mistaken for pustular and gummatous syphilis!) The skin foci rupture and produce irregular ulcers with pus covered edges. See Zieler, concerning chronic glanders.

Actinomycosis of the skin is most frequent in the cheeks and the neck, secondary to mouth infections; on the trunk, particularly the back, secondary to vertebral, rib, or lung involvement; on the abdomen and the inguinal region, secondary to intestinal actinomycosis; (p. 466 and forward, also pp. 557, 558 and 1145). Primary cutaneous actinomycosis is quite rare, usually occurs on non-covered parts (Vignolo-Lutati,

lit.), and is observed following injuries (Marcus), as a chronic, sometimes syphilitic-like, ulcerating process, sometimes lupus-like with nodules, and no tendency to spontaneous healing (Lieblein). A disease, formerly believed actinomycotic, has lately been found due to another fungoid irritant, the discomyces Thibiergi (Bostroem-Israel) and described by Ravaut and Pinoy as a new form of skin discomycosis.

When the cicatrizing fistulous growth spreads to the bones and joints of the foot and thigh, the lesions resemble **Madura foot** (mycetoma pedis), (cases of Tusini and Bollinger). (Illustrations, among others, by Bockenheimer.) The latter disease is endemic in the island of Madura, close to Java, in India, and other tropical countries, but has been seen isolated in Europe. It progresses slowly with marked swelling of the foot caused by a fungus similar to actinomyces (Kanthak), but without its basket-like crown; the organism enters the skin through minute abrasions. Bright yellow and black bits of fungi similar to actinomyces are discharged in the thin, fluid, evil-smelling pus. According to Caminiti, streptothrix rubra (Vincent) is the cause, but the etiology is not uniform (Reynier et Brumpt). Lit. by Babes, Oppenheim, Dubenhofer, Koch and Stutzer, Pepere (concerning black Madura foot), Babes, Schmincke, Catsaras, Miescher (comprehensive lit.), mycetoma pedis nostras, produced by a new pathogenic streptothrix, with remarks on the classification of trichomycetes (streptothrix, actinomyces); Vuillemin, classification of mycetomas, with the formation of granules, local tumor-like or ulcerative processes brought about by various kinds of fungi.

The Skin Diseases Produced by Fungi; Dermatomycoses

Mycoses Confined to the Skin.— Newer investigations, particularly those of Bloch, make it apparent that the dermatomycoses can no longer be looked upon as simple, or purely local skin diseases, but as involving the entire body and calling forth reactions, particularly along immunological lines, in most of the cases. These questions are discussed in the work of Bloch concerning the general pathological importance of the dermatomycoses. See also Kusunoki, E. Sutter (lit.), concerning immunity and hypersusceptibility in trichophyton diseases, and the general references of Riecke.

(*a*) **Favus** (tinea favosa) is a contagious disease, particularly of the scalp in children, characterized by rounded, sulphur yellow discs with central depressions, through which a hair protrudes. They reach the size of lentils to ten cent pieces, and are covered with crusts with a mole or mouse-like odor. Invasion follows the hair into the follicle (favus-scutula).

The scutulum (1–1.5 mm. thick), still covered by thin epidermis, is examined in water, glycerin, or lime water to disclose threads of fungi

and conidium spores, both very polymorphous, and mixed with debris, pus cells, and micrococci. According to Unna, it is a fungus body which has entered the horny layer, the perpendicularly ascending fungus threads extending out horizontally in the skin. The cause of favus is the achorion Schoenleinii (discovered 1839; the first fungus obtained from humans; fig. 1024). It is a mycelium composed of threads (hyphen), partly jointed and partly unjointed, with many right angled side buds. Spores appear at the ends of the threads and produce rosary-like chains; there is more mycelium than spores.

When the covering of epidermis is washed from the scutulum, the dry, yellow white, mortar-like masses of favus become visible; when the scutulum is removed in vivo, it reveals a red moist surface. Favus long continued, destroys the hair in the involved situations, for the fungi invade the hair shaft, the root, and the sheaths. The hair is lustreless, dusty, falls out readily, or breaks when above the epidermis.

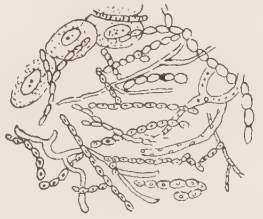


FIG. 1024. — Achorion Schoenleinii from a favus-scutulum; mycelia and spore chains; above left, three epithelial cells. (High power.)

The hair of favus is not broken off, the trichophyton hair is broken off short, the microsporon hair is broken off longer, and the hairs always point in the same direction. There is more or less marked infiltration of the corium.

The fungus attacks the eyebrows, situations covered only with down, and also the nails. The latter are infiltrated by sulphur yellow inclusions, undergo caseous degeneration, become crippled, lustreless, and cloudy (onychomycosis favosa). See Talat (illustrations), and Plaut, (lit.), concerning universal favus.

Quincke and Unna assumed that various kinds of fungi are responsible for these diseases; others, such as Pick, Waelsch, Scholtz, etc., believe, on the other hand, that there is a polymorphous development of a single fungus, the achorion Schoenleinii.

Newer investigations have identified a number of varieties of fungi: (1) achorion Schonleinii, carried from human to human, favus of the human type; (2) fungi of the animal type, among them (*a*) achorion Quinckeanum, (*b*) achorion gypseum Bodin and (*c*) achorion violaceum Bloch; these are transmitted to humans, chiefly by mice, as demonstrated first by Quincke in the case of the achorion named after him (a-fungus). The animal types, however, never produce typical scalp favus, but always a relatively acute and benign lesion of the smooth skin, the eyebrows or the beard, resembling the diseases caused by the trichophytons (sycosis and kerion Celsi). See Bloch (lit.), Buschke and Michael, (lit.); see Kaufmann Wolf concerning the demonstration of pathogenic blastomycetes.

The infectious skin disease produced by the **trichophyton tonsurans** is modified according to whether it attacks situations containing hair or not. The trichophyton fungus, of very fine threads with few branches, and few conidia, produces no scutula. According to Gruby, Sabouraud and others, and the investigations of Krosing and Halgand, they are divided into a number of groups. These fungi grow from the follicles into the substance of the hair (endothrix forms—the human type), or around the hair like a sheath (ectothrix form; in the animal type, see Bloch); *i.e.*, in the earlier stage, the hairs are infiltrated by threads (quite similar to achorion); in later stages they are surrounded by rounded, or elongated, oval, usually chain grouped, conidia or spores. In situations without hair, the fungi lie in the deeper layers of the stratum Malpighii.

The group of trichophytons of human origin, *i.e.*, the endothrix types, may be differentiated into: trichophyton crateriforme, acuminatum, violaceum; whereas in the trichophyton forms of animal origin, *i.e.*, ectothrix types, there are the trichophyton gypseum (with eight subtypes), trichophyte felineum, and others (see Chajes).

Herpes tonsurans (trichophytia superficialis) on non-hairy places, appears as vesicles (herpes tonsurans vesiculosus), or red desquamating spots, discs and circles (herpes tonsurans maculo-squamosus). Herpes tonsurans vesiculosus is comparable to herpes circinnatus, and is characterized by annular forms composed of isolated vesicles on a reddened base, with desquamating centers. Herpes tonsurans maculosus et squamosus appears by preference in the borders of the hair and the nape of the neck as penny to dollar size, red, desquamating, and disappearing, annular formations becoming pale under pressure and developing from their centers toward the peripheries. But it may be a generalized acute eruption on the trunk and on the extremities.

Trichophytia unguium, onychomycosis trichophytina, is caused by the trichophyton in and under the nails producing cloudiness, nodular thickening, lamination, and brittleness.

Herpes tonsurans capillitii (trichophytia profunda) shows rounded bald spots in the scalp as large as dollars, in the center of which the hair is dry and brittle. Later, merely the stumps of unequally broken hairs remain covered by scales. The edges of the tonsur are red. Entrance into the hair follicles and vigorous inflammation in the surroundings causes pustules, crusts and abscesses with granulation tissue containing many giant cells and sharply outlined foci of epithelioid (tuberculoid) cells, even in the subcutis. The demonstration of fungi in sections is very difficult. Neighboring bald spots may coalesce until the greater part of the scalp is bald, but healing can occur before this is complete. When the organism spreads deeply into the skin and produces generalized skin

symptoms in various forms such as eczematous, lichenoid, nodose and other eruptions (Guth, Arzt and Fuhs), the condition is known as "trichophytidia" or "trichophytosis."

Sycosis parasitaria is a trichophytia profunda in the beard. Beginning as a superficial lesion, it soon becomes deep and is accompanied by vigorous inflammatory processes. More marked inflammation of the follicles produces nodules, pustules, abscesses, scabs and doughy, tumor-like, very soft, papillary overgrowths, through which project stumps of hair. These can easily be pulled out, and their roots are surrounded by a white gray or a yellow gray sheath. The disease lasts for weeks. Healing usually ad integrum. Only rarely is it combined with akerion Celsi.

Microsporia is an endemic infectious disease very frequent in certain countries such as England, France, Belgium, but also in Switzerland and Germany, Vienna (His, Plaut, Glaser, Chajes concerning epidemics in Basle, Hamburg, Berlin, Schoneberg, Braunschweig), in school children, usually boys. Its chief characteristics are rounded, gray, desquamating spots in the scalp with sparse, broken off hairs. Inflammation is practically never present. Under light pressure on a cover glass the mosaic-like microsporia hair sheaths break up into isolated spores (in trichophytia also into chains of spores). There are differentiated: a microsporon Adouini, the chief representative of the human type, and the less feared, inflammation producing, and much more readily healing, microsporian, carried by animals; thus, the microsporon canis s. lanosum, the microsporon felineum and others; in infections by the latter, the spread over the trunk is greater, more frequent, and usually in the form of rings. See Plaut and Chajes concerning the microscopic details, culture methods, etc. The Audouin form is very chronic; spontaneous healing has often been observed at puberty. (For x-ray treatment, see Sabouraud, Berger and Bloch, lit., Stein, Sternthal and others; see also lit. by Buschke and Michael.) The microsporia has its counterpart in a highly contagious, stubborn trichophyton disease of the surface of the scalp in children, "teigne tondante trichophytique," known in the schools of the larger cities, particularly Paris and London, for many years, and caused by various endothrix forms of the trichophyton (crateriforme, acuminatum, violaceum).

Eczema marginatum (Hebra), according to Kaposi and others, belongs to herpes tonsurans. It is found particularly often on the scrotum and the neighboring skin, the thighs, but also on the trunk and extremities (Buschke). It is characterized by small nodules and vesicles with irregular peripheries on a brown pigmented, and centrally pale, skin area. Newer investigations, however, put this disease in a distinct class; the cause is said to be the epidermophyton inguinale (Bloch, lit., Sabouraud).

Tinea albigena (Niewenhuis), a chronic disease involving most often the palms of the hands and the soles of the feet, produces symmetrical papules and vesicles, with superficial dermatitis; it belongs to the group of the trichophytons. The final scars lead to the characteristic discoloration which gives the disease its name. It may also attack the nails, the glans penis, and the internal leaf of the prepuce (Helm).

Tinea imbricata is also classed with this disease; it appears in epidemic form, chiefly in the South Sea Islands. This fungus is believed by some to be related to the trichophytes, but according to others, is a true aspergillus (Wehmer); it grows in the rete Malpighii. The epidermis displays annular figures, and is separated from the cutis in small lamellae. The edges remain adherent around the periphery, and thus look like the shingles of a roof; hence the name, imbricata. As a rule, the skin of the entire body is involved with the exception of the face. (Good illustrations by R. Koch.) For more details of tropical trichophytosis, see Castellani.

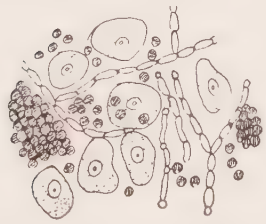


FIG. 1025.—Microsporum furfur. (High power.)

Pityriasis versicolor, caused by the microsporon furfur (fig. 1025), usually occurs on parts of the body covered by clothes, and those more prone to perspiration, thus the back, the abdomen, the contact surfaces of hanging breasts, the scrotum, and the upper parts of the thighs. Yellow, or hyperemic red, to brown red points and spots extend over large areas of the skin in irregular map-like outlines; the skin is covered with delicate clay-like scales.

Microscopically, the removed scales (epidermis lamella), show criss-cross, broad, whorled, mycelial threads, with little tendency to branching; between them are large groups of spherical, markedly refractile, spores (fig. 1025).

This condition heals with difficulty and readily recurs, usually disappears after many years; in older persons it can no longer be found (Kaposi), and is seldom seen in children.

Concerning pityriasis maculosa chronica, or parapsoriasis en plaques, called by Brocq, erythrodermia pityriasique en plaques disseminees, see the comprehensive work of Arndt (lit.). Neither fungi nor bacteria have been found in the fine scales. See also p. 2098.

In **erythrasma,** glistening or fatty spots appear on and near the surfaces of contact of the scrotum and skin of the thighs, the folds of the breasts, etc.; they are red yellow or brown, and itch under the influence of perspiration; the microsporon minutissimum is found (v. Barensprung), similar in appearance to microsporon furfur, but all the structures are much smaller and more delicate.

Generalized Mycoses

Thread Fungi (Hyphomycetes).—Sporotrichosis is produced by the sporotricon de Beurmann (1903), and others such as the sporotricon Schencki. The lesions are at times similar to those of tertiary lues, at other times similar to tuberculosis. The disease is of considerable practical importance. Human sporotrichosis is found chiefly in France, animal (horse) sporotrichosis, in certain regions of the United States, thus in Pennsylvania, (K. F. Meyer). According to de Beurmann and Gougerot, three clinical forms may be differentiated: (1) the most frequent is sporotrichum cutanee gommeuse, in multiple disseminated foci with tough nodules in the cutaneous and subcutaneous tissues, becoming larger and larger till they reach the size of hazel nuts or larger, adhere to the skin, become softened in their centers, and then perforate over broad areas (like gummata); when they disappear earlier they leave behind a large pigmented spot. (2) Another form described as lymphangitis sporotrichotica gummosa shows a primary lesion in the form of a sporotrichotic chancre on one extremity or in the face; tough strands radiate from this along which larger nodules are formed. It can hardly be differentiated clinically from analogous tuberculous conditions, a fact also true of other tuberculoid sporotrichoses, but glandular swellings are usually absent (Dind), and the polymorphism of the symptoms is much greater in sporotrichosis. (3) Extracutaneous sporotrichosis may appear in various organs: muscles (gummata and abscesses), mucous membranes (mouth, pharynx, see p. 621, nose), eyes, (eyelids, conjunctiva, cornea, iris, etc.), the tendon sheaths, and particularly the bones, perhaps isolated, as in the tibia, as spina ventosa of the phalanges, the joints, etc., (p. 1147); it is easily mistaken for lues or tuberculosis, the more readily, since sporotrichosis frequently develops in conjunction with an old tuberculosis. Histologically, the tissue is a characteristic mixture of tuberculous and syphilitic-like structures, but with little necrosis and much suppuration (details by Arndt). For diagnosis, cultures may be tried on maltose agar kept at room temperature (not under 15°C.), whereby, in four to six days, there are presented characteristic microscopic masses of dense straight or bent, branched threads, to which are attached innumerable pedunculated ovoid spores. Dextrose peptone agar is also a good culture medium (Gougerot). Intraperitoneal injections into male rats produce typical epididymitis and funiculitis sporotrichotica in two to three weeks; all organs may be attacked.

Sporotrichosis may be a local condition, or may be accompanied by general symptoms of fever and cachexia, when the fungi are carried through the blood stream; it may even produce death. The therapy is by

means of iodine. (Lit. by B. Bloch, and to 1910 by Arndt; see also Kren and Schramek, de Beurmann et Gougerot (lit.), Hamburger, Hecht.)

See Gougerot and Caravin concerning hemisporosis, a new human mycosis, which macroscopically and microscopically is quite similar to tuberculosis, and is produced by the "hemispora stellata vuillemin." See also p. 1147 under bones.

See Bloch and A. Vischer, concerning other, rarer, dermatomycoses, such as oidiomycosis, producing multiple cutaneous and subcutaneous ulcerating furuncle-like nodules; also the rarer cladiosis, producing multiple verrucose and papillary skin foci important in differential diagnoses.

Budding or yeast fungi (blastomycetes):

Blastomycosis is a chronic skin disease, rare in humans, produced by blastomycetes. It takes the form of a nodular folliculitis with central destruction, and subsequent flat cicatrization; new rosary-like nodules spread in the neighborhood; another form produces tumor-like infiltrates, becoming deep, crateriform ulcers of gummatous appearance (Haberfeld), with overhanging edges and gray or brown secretions containing the fungi.

Occasionally, very extensive papillomatous growths are produced resembling lupus papillaris. Since the regional lymph nodes are attacked, their neighborhood infiltrated, and the skin ruptured, the disease may be mistaken for sarcoma or carcinoma.

The internal organs such as the lungs, kidneys, spleen, and the bones, contain metastatic tumor-like granulation foci or abscesses (p. 1147). When the distribution is thus generalized, the course is usually fatal (see the case of Verse). The granulation tissue is extraordinarily rich in giant cells; the reaction of the epithelium is marked; in fact, it grows like an epithelioma, but is destroyed by abscesses. The organisms are usually easy to demonstrate. The tendency to connective tissue new growth is very small in contrast to actinomycosis.

To demonstrate these fungi: stain with very weak Ziehl-Neelsen's carbol fuchsin, or Gram's method and counterstain with fuchsin; culture in sweet wort, or on carrot media streaked with lanolin (v. Petersen). Busse (lit.), and Buschke succeeded in growing them in pure culture from human inflammatory foci, and have demonstrated their pathological importance by experimental inoculation (mice). See the illustrations and lits. by Ricketts and Finger, da Rocha-Lime, Stober.

See Stein, concerning the similar blastomycosis americana (Gilchrist's disease). For blastomycetes and malignant tumors, see Leopold, Roncali, (lit.) and compare contrariwise Sternberg. Systematic pathogenic and parasitic blastomycetes, and lit. by Sakagawa. [For yeast infections of the skin, see Greenbaum, S. S., and Klauder, J. V., Arch. Dermat. & Syph. 5: 332: 1922. For ring worm culture media, see Weidman,

F. D., and McMillan, T. M., Arch. Dermat. & Syph. 4: 451: 1921. For blastomycetoid bodies in sarcoma-like tumors, see Weidman, F. D., and Douglas, H. R., Arch. Dermat. & Syph. 3: 743: 1921.—*Translator.*]

Skin Diseases Produced by Animal Parasites (Dermatozoonoses)

Among these parasites, two groups may be differentiated (Kaposi): 1. such which dwell exclusively, or for a certain length of time, on the human skin, *i.e.*, true parasites, dermatozoa, consisting of the itch mite (acarus scabiei), the demodex or acarus folliculorum, sand fleas (leptus autumnalis), the wood borer (ixodes recinus), etc. To this group may be added the echinococcus and the cysticercus, which live in the subcutaneous tissues; and also, according to the opinion of certain authors, the "cause" of epithelioma molluscum; 2. those which remain only for a time on the skin to obtain their nourishment, but otherwise live close at hand (hair, clothing), the epizoa. These are lice (head lice, pediculi capitis; body lice, pediculi vestimentorum; and pubic lice, pediculi pubis); fleas, (pulex irritans), bedbugs (cimex lectularius), flies, and other insects.

See Lehner (lit.), concerning the occurrence of the larvae of gastrophilus equi, probably carried by the two-winged gastrophilus equi, and invading the skin as larvae migrans, and producing vesicles and itching (creeping disease).

The importance of the parasites consists, in part, of direct damage, and, in part, indirect damage through itching and scratching. In addition, they act as carriers of dangerous pathogenic organisms, *e.g.*, bed-bugs as carriers of trypanosomes, body lice as carriers of spotted fever, etc. See lit. by Mollers, Sangiorgi, Hoffmann, Sikora, Hase, Heymann.

Scabies or itch is a contagious, itching and scratching, skin disease, produced by the presence of the acarus scabies (sarcoptes hominis) in the epidermis, as the turtle-like itch mite which reaches the size of the head of a pin.

The male mite (0.25–0.3 mm. long, 0.15–0.2 mm. wide), is usually present in less number than the female, which is about twice as big; they do not bore true passageways, but remain in soft furrows in the epidermis and in the neighborhood of the passageways bored by the females. The pregnant females enter the horny layer in horizontal or oblique directions (never at right angles), and remain in the horny layer (Torok, Unna), where this is thick; where the horny layer is thin (Schicha), they reach to the depths of the rete. The passageways thus produced have a length of 1 to 3 centimeters or more, and in them the females deposit their oval eggs (20–50 or more) and their excrement. Their course is tortuous, somewhat raised, and the end where the mite reposes, has a small button-like prominence. In about six days, six-legged larvae hatch from the

eggs, reach the surface of the skin through the so-called tunnels, in which they then bore a small short nest, and reach sexual maturity in a few weeks, after undergoing three different processes of covering (after their first covering they have four pairs of feet). The sexually mature mites then burrow new passageways for themselves. The danger of contagion from a pregnant female is particularly great in the warmth (bed), when the mites begin to burrow, producing the itching, and to wander to the skin surface. In cool surroundings, as during the day, the animals are quiet. In damp surroundings, the ova and the mites live about four weeks, even separated from a human body. Air heated from fifty to seventy degrees kills the animals in about three quarters of an hour. [Caution must be exercised in examining and measuring ova, etc.; pressure on the coverglass, *e.g.*, will produce differences in appearance, etc.; see Weidman, F. D., J. Parasit. 6: 155: 1920.—*Translator.*]

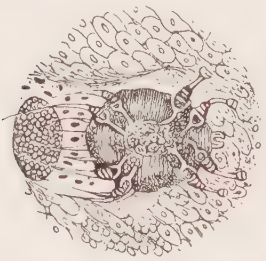


FIG. 1026.—Female acarus scabiei with one egg in a tunnel in the epidermis; somewhat diagrammatic after Kaposi.

From direct irritation of the mites, and from the itching and scratching, various kinds of efflorescences are produced, such as eczematous papules and pustules, which, after scratching, are covered with scabs, hemorrhages and excoriations (histological by Volk).

The chief foci of the itch burrows are found in situations with tender skin, such as the lateral surfaces of the fingers, the inner surface of the root of the hand, the extensor surfaces of the elbows and knee joints, the nates, the axillae, the feet between the toes, and the back of the foot.

Molluscum contagiosum, (epithelioma contagiosum, epithelioma molloscum), according to the debated opinion of many authors (Bollinger, Neisser and others), is a tumor-like formation, probably contagious, produced by coccidia. The white, shimmering, waxy, glistening, almost transparent, rounded, warty or pox-like structures with central depressions are of the size of the head of a pin to peas, similar to those of varicella.

They are fairly numerous, as isolated nodules or aggregate groups, particularly in children, on certain situations such as the penis, scrotum, trunk, labia, perineum, flexor surfaces of the extremities, the face, the neck, the nape of the neck. They remain weeks, months, or years, and may disappear spontaneously. They do not itch, and are painless.

On section, the molluscum (fig. 1027), can often be pressed out in toto from its bed and shows a lobulated structure, similar to that of a hyper-

trophic sebaceous gland, inasmuch as cell masses coming from the rete, are separated from each other by fibrous septa. The papillary bodies in the cutis are compromised, the latter is applied like a capsule around the molluscum. In their centers is a sort of cavity, in which the tags or the lobules touch each other, and in which repose "bodies" and cornified cells.

According to the parasitic theory, the formation of the epithelial cell masses is provoked by the parasites (protozoa), which invade the rete cells, whereupon there is enlargement in lobules of the preformed rete depressions. The parasites first produce small protoplasmic bodies in the epithelial cells, they then become nucleated, and later occupy the entire epithelial cell while the nuclei undergo division and enlargement. The nucleus of the cell, pushed toward its wall, gradually disappears, while the cell early acquires a bordering membrane surrounding the parasite, as it becomes divided into a large number of finely granular structures. The parasites first lie in the epithelial cells of the isolated lobes, but the epithelial cells soon shove them toward the middle of the cavity of the molluscum. There they lie between flat epithelial cells, fat droplets and fat crystals, as large, egg-shaped, anucleated, glistening bodies, uncovered or surrounded by a covering of epidermis, as the so-called molluscum bodies. Juliusberg discusses the filtrability of the virus of human molluscum contageosum. O. Israel, Torok and Tommasoli, Unna, Kromayer, Saul, Scherber (lit.), Cedercreutz (lit.), and others, believe the parasites are homogeneous products of a degenerative process in the epithelial cells, *i.e.*, "dyskeratotic" superficial cells, the peculiarity of which is produced by a particular property of the virus (Jadassohn, lit.); see also Libschutz, who described "elementary bodies," Leeben, Kreibich, v. Prowazek, Leber and da Rocha-Lima. Sabella and others consider them particular molluscum parasites, whereas Sanfelice believes the transfer to pigeons was by toxins obtained from diseased epithelial cells. Knowles saw epidemics in families.

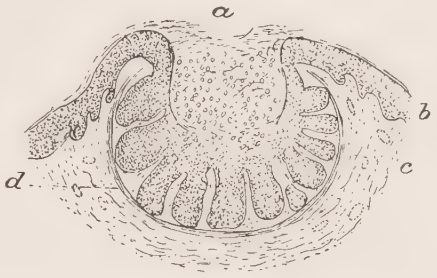


FIG. 1027.—Molluscum contagiosum; in center, the gland-like epithelial proliferation within which lie the molluscum bodies; above at a, is an apparent efferent duct with cornified epithelium and "bodies;" b, epidermis; c, connective tissue of cutis; forming at d, a sort of capsule around the molluscum (half diagrammatic).

Darier's disease (psorospermosis folliculaire vegetante), a particularly chronic dermatosis, consists of small, dirty yellow gray or brown crusts and scabs as tightly adherent outgrowths. More rarely, there are larger, moist tumors. The process is a keratosis and parakeratosis of the hair follicles, and hair shafts; the process begins in the epidermis, pushing the

rete deeper into the less infiltrated corium; it then passes to the openings of the glands, which are dilated and filled with incompletely cornified cells. Thus the condition is spoken of as keratosis follicularis. In the epithelium of the cornified areas, Darier found homogeneous, markedly refractile, "corps ronds," which he erroneously believed to be psorospermia; these are said to infect the skin, and so produce epithelial overgrowth with marked cornification, and superficial inflammation of the cutis. Others, such as Buzzi and Miethke, Boeck, Unna, Kroesing, Petersen, Bukovsky, believe the "bodies" are keratohyalin and eleidin, substances which develop in cornified cells, or hydropic bodies appearing during the pathological cornification process (O. Sachs). Lit. by Bizzozero.

Oriental boil or Aleppo boil (tropical ulcer, Delhi boil), is a disease observed in warm countries, probably communicated through the bite of an insect (endemic boils); it begins as a papule, from which is formed a pustule, and then an ulcer which may reach the size of a dinner plate, with soft rims, undermined edges, and diminished tendency to cicatrization (torpid). In weeks, or many months, the indolent ulcer may finally heal leaving radiating scars (may be mistaken for lues!). These boils may be multiple primarily; thus de Beurmann observed twelve boils on the legs. It is chiefly a granulation tissue spread in the cutis, and undergoing suppuration; in the pus and tissues are large numbers of protozoa (trypanosomes), similar to the Leishman-Donovan bodies of kala-azar (p. 236); Wright calls them helcosoma tropicum, Nattan Larier and Nicolaides, piroplasma tropica, Marzinowsky, ovoplasma orientale, Reinhardt (lit.), Leishmania tropica. De Beurmann (lit.) proposes, with Laveran, the term leishmaniosis ulcerosa cutis rather than oriental Aleppo, Kiskra, Delhi or Bagdad boil and others. The activity of these bodies stimulates the focal collection of peculiar macrophages, but the tissue of the boil also contains many lymphocytes, plasma cells, and giant cell-containing nodules in the deeper layers (Reinhardt). The disease has been transmitted to apes and to white mice (intraperitoneal, intravenous, and oral). Cultures reveal flagella forms. See also Reinhardt, Kuhn (lit.), Bettmann, v. Wasielewski, Arndt and Zurn, and particularly Schrotter, Felke.

See p. 211, and Gery, concerning the trypanids in trypanosomiasis, (p. 2141), some of which resemble syphilitic dermatoses, some dourine (mal de coit of the horse), in the form of roseolar, macular, cercinoid, serpiginous, and even nodular erythemas.

Abnormal Pigmentations, and Lack of Pigment

Abnormal circumscribed or diffuse pigmentation of the skin is classified as: (1) an increase of the normal autogenous pigment of the rete cells and the upper layer of the corium; (2) a deposit of blood or bile pigment

(p. 1010); (3) coloring matter introduced externally (tattooing, p. 254), and other pigmentations coming from the blood stream.

Among the latter may be mentioned argyria, produced by the continuous internal use of silver salts. The silver is deposited in finely granular or diffuse precipitates in the cutis, in the membrana propria of the sweat glands, thickly under the rete, or in the upper part of the sebaceous glands (not in the rete, the epithelium remains free and is unstained, in contrast to the pigmented cutis). All light parts of the skin are dark gray, bronze-like, blue gray, shimmering, to black brown. According to Kanitz (lit.), the silver precipitate is carried by the elastic fibers (see also Dohi, Kino), and the fixed bordering membranes (membranae propriae), connective tissue around the hair follicles, connective tissue walls of the sebaceous glands, perimysium and perineurium. In the internal organs (kidneys, p. 1382), the silver lies only in the connective tissue (Riemer). Local argyria may appear on the hands of workers in silver, or after application of lunar caustic.

According to their etiology, various pigmentations are recognized:

Among the first are those which appear at birth, at least are congenital (and hereditary) local pigmentations, in the form of tumor-like, richly cellular structures, known as birth marks (naevi pigmentosi) or lentigines, or ephelides, or xanthomas, or xanthelasmas.

Their histological structure is concerned and will be discussed more fully under tumors, but their gross anatomical pictures may be sketched as follows:

Naevi pigmentosi are congenital, brown or black marks (birth marks, naevi materni), with flat (naevi spilus), or prominent (naevi prominens), or warty (naevi verrucosus) surfaces, those of the latter perhaps thickly strewn with hair (naevi pilosus). Pigment naevi are usually small, but occasional hairy naevi may spread over large areas of skin (hairy humans).

Lentigines are manifest only after birth, reach the size of heads of pins or lentils, and never disappear; they belong to the soft warts (p. 2187), and show heaps and strands of pigmented cells, just as these.

Ephelides, freckles, are non-prominent, small, yellow brown spots accentuated on uncovered situations under the influence of sunlight, but also appearing on covered situations, chiefly in individuals with rosy skins; they appear and disappear, or persist. (See Hammer, Meirowsky, concerning their hereditary nature, possibly due to germinal anlage, at first latent, and then appearing under the influence of light in areas with a tendency to pigmentation.) Microscopically, the basal cells, perhaps also the higher layers, show more melanotic pigment and more melanoblasts in the papillary bodies, but cell masses and strands are lacking.

The blue, so-called Mongolian birth marks (Balz), appear at the upper end of the rima ani toward the back; they reach the size of a silver dollar,

have smooth surfaces, without ectasy of vessels, and consist of large spindle and stellate pigment cells in the deeper layers of the corium. In spite of isolated exceptions, Schramek (lit.) believes they are indications of an admixture of the yellow (Mongolian) race. But they also occur in the white race. W. Fischer believes they are atavistic, a remnant of monkey ancestry.

The arcus senilis, or gerontoxon, is a symptom of old age manifested in the cornea as a light gray circle around the periphery, according to Kawamura, due to an infiltration of doubly refracting substances (cholesterin esters) in the cornea.

Xanthomas (xanthelasmas) occur as the frequent xanthoma palpebrarum (*a*), occasionally inheritable, and localized to the skin of the

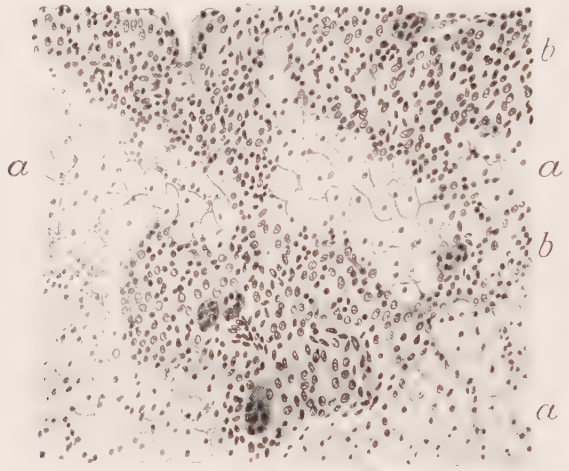


FIG. 1028.—Xanthomatous giant cell-containing sarcoma from a lobulated tumor of the heel as large as a hen's egg, extending to the periosteum; a, xanthoma cells (contain doubly-refracting substance); b, polymorphous sarcoma cells with giant cells; man 34 years old. (High power.)

eyelids, as sharply outlined yellow spots or nodules; or as the rarer xanthoma multiplex (*b*), or xanthomatosis, or "generalized xanthomatosis," occasionally familial (Arning), in which the yellow and brown spots, nodules or tumor-like papules, are scattered in single or numerous examples, (xanthoma planum, or tuberosum). The nodules vary in size from that of millet seeds to lentils to beans, and prefer the extensor surfaces of the arms and legs, *i.e.*, along the elbows and knees. Some cases also develop coincident tumors in the tendon sheaths of the hands (Torck, lit.). They are composed of collections of peculiar large cells, wavy and foamy in alcohol preparations, while, in formalin frozen sections, they show droplets and needles of a doubly refracting lipoid substance (protagon), or cholesterin esters (Stoerk, Pinkus and Pick, Pringsheim,

Kawamura, and lit. in appendix). That infiltration with neutral fat (singly refractile) occasionally occurs, perhaps in earlier stages, is shown by the case of Fahr. A hypercholesterinemia, or a general cholesterinsteatosis is often considered obligatory for the origin of xanthoma in all forms, (see, for example, B. E. Schmidt), or the "xanthelasmatic" diseases, (Aschoff). It may thus be considered an example of a diathetic dermatosis (Bloch, lit.), but not in all cases, since in those studied by Siemens (comprehensive lit.), and Rosenthal-Braunisch, the extensive xanthomatous infiltration of the skin was accompanied by a normal blood cholesterin content. But hypercholesterinemia is a symptomatically important accompaniment in a goodly percentage of cases. It must be doubted that long continued lymph stasis, assumed of importance in local xanthoma, really plays a role in addition to a hypercholesterinemia, (Spiess, lit., Hoessli, Lubarsch). The xanthoma cells are both overgrown fibroblasts and overgrown lymph vessel endothelial cells both infiltrated by lipoids; the former are also called macrophages (p. 9); but, according to others, the macrophages are particular cells of the connective tissue belonging to the reticulo-endothelial apparatus (p. 219). It is difficult to determine their genesis in every case. Xanthomata multiplex occurs (1) idiopathically, (2) symptomatically, *i.e.*, related to disturbances of cholesterin metabolism, thus of the liver, with icterus (Chvostek), nephritis, some tuberculoses (Sikemeier), diabetes, etc., facts of possible diagnostic importance. In fact, diabetics frequently have so-called xanthosis (v. Noorden) or diffuse xanthosis (xanthochromia), a yellow discoloration of the skin without the formation of nodules. According to Umber, every xanthosis is related to disturbed intermediate metabolism and lipoidemia; Fahr believes lipemia and xanthosis are the expression of diminished lipolysis, and failure of metabolism of fat. The canary yellow coloring matter of xanthosis is said to be a lipochrome derived from the food (Umber, Burger and Reinhart, Salomon, lit.). In such cases there are apt to be xanthoma structures in the internal organs, *i.e.*, nodules of large overgrown foamy cells as in generalized xanthoma, or xanthoma of the eyelids (Lubarsch, Bross). Diabetic, icteric and nephritic xanthoses are often transitory, (Chvostek speaks of xanthelasmias), as a differentiation from the xanthomas which are tumors of the lipoid substance group (Aschoff, Chvostek). Xanthomas en tumeurs (*c*), are neoplasms of a peculiar kind, lobulated, tough, elastic, some as large as hens' eggs, on section variegated, gray, transparent and light brown with characteristic cloudy white foci produced by numerous xanthoma cells. Their sites of preference are in the knees, the calves, and the elbows; they grow from out the subcutaneous tissue, and may adhere to the underlying tendons, to the periosteum, or to the joint cap-

sules. The author saw such a tumor (xanthomatous giant cell sarcoma) situated intracutaneously in the neck of a one and one-half year old girl. They are quite rare. The histological structure varies; in addition to the characteristic xanthoma cells which show doubly refractive needles and granules in polarized light, they consist of polymorphous sarcoma cells, and multinucleated giant cells; we have repeatedly found iron-containing cells (Kammer). Low saw xanthoma tuberosa multiplex with foci in the tendon sheaths, and probably also in the heart. But, as a general rule, these tumors produce only local recurrences, do not erode blood vessels, produce no metastases, and can therefore not be identified with true malignant sarcomas. They are sometimes identified with lipoid infiltrations, (true fibro xanthosarcomas of the adrenal region, with numerous metastases, were described by Dietrich, Noethen). See also p. 2064 under tumors of the tendon sheaths. Corten (lit.) described a xanthoma which he believed was of epithelial origin coming from misplaced germinal sebaceous gland tissue. According to Kirch, true blastomas of all sorts and localizations may occasionally contain xanthomatous, *i.e.*, foamy cells. Even so, the author believes a particular classification is necessary for the xanthomatous giant cell-containing sarcomas established in dense connective tissue in preferred localizations; he does not believe, as does Fleissig (lit.), that they are like tendon sheath tumors, *i.e.*, a particular kind of granulation tumor, (see also Petri for foamy cell tumors). [For xanthoma diabeticorum, see Major, R. H., J. Hop. Hosp. Bull., 35: 27: 1924. In two recent cases, the translator saw the multiple skin lesions disappear in about two months under insulin therapy. For xanthomas in general, see Bloodgood, J. C., Garrett, C. A., and Smith, D. T., Arch. Surg., 8: 882: 1924. For xanthoma tuberosum with lesions in the central nervous system and other tissues, see Weidman, F. D., and Freeman, W., Arch. Dermat. & Syph., 9: 149: 1924. For xanthoma tuberosum multiplex in childhood with visceral and tendon sheath involvement, see Knowles, F. C. and Fisher, H. N., J. A. M. A., 77: 1557: 1921.—*Translator.*]

Chloasmas are yellow or brown spots with smooth surfaces of varying sizes and shapes. They occur under both physiological and pathological conditions, thus in women during pregnancy (chloasma gravidarum on the forehead, the alae nasi, the cheeks), or in the female genital apparatus in various diseases (chloasma uterinum). These symptomatic chloasmas disappear at the time of the climacterium.

In chloasma gravidarum there is thought to be a relative insufficiency of the adrenal medulla.

Chloasma cachecticorum describes brown spots similar to those of chloasma uterinum, or diffuse dark discolorations of the skin appearing occasionally in cachectic individuals (phthisics, carcinoma, alcoholics,

vagabonds, etc.). Some cases of this kind are similar to the severest degrees of bronze pigmentation (p. 1283).

A further group is caused by thermic, toxic, or traumatic influences directed to the skin.

Chloasma caloricum is the transient, brown discoloration of the skin produced by the rays of the sun, although raw, cold winds may have the same effect. Chloasma toxicum is caused by the application of certain irritant substances such as mustard plasters, cantharides plasters, etc. This type of pigmentation often remains for life. Chloasma traumaticus is a brown to black pigmentation of diffuse extent or punctiform or striped arrangement produced particularly by scratching. In itching skin diseases, particularly those brought about by certain types of animal parasites, this chloasma is practically always present, but it may appear from other causes, *e.g.*, too tight lacing. Many use the name melasma for this dark pigmentation. The discoloration occurs from granular precipitation of blood coloring matter and diffusion into the tissues. It is often found in eczematous thighs. Removal of the pigment and lodgment in the regional lymph nodes (usually the inguinal), is frequently observed (Jadassohn).

See p. 1283 under adrenals, concerning the bronze skin, melasma suprarenale, of Addison's disease (Heudorfer); see p. 926 concerning bronze diabetes.

Melanosis of the skin, a brown pigmentation of the otherwise unchanged epidermis, follows the internal use of arsenic (Schucany, lit.); it disappears in weeks or months. See p. 2195, concerning the melanosis of the skin and the internal organs in melanosarcomatosis.

Xeroderma pigmentosum (Kaposi, Lukasiewicz, Matzenauer, Lowenbach, Adrian, lit., Rouviere, lit.), is debatably caused by pathological congenital anlage, possibly from a summation of various pathological germinal disturbances occurring in certain families (lit. Schonnefeld); as a rule, it consists of the sudden appearance of local infiltrating foci in earliest childhood, and usually, even though not exclusively, on situations exposed to the light, such as the face and hands. At first, there are red spots which are quickly pigmented with brown, and which reach the size of lentils to peas, or larger, from coalescence. It is also thought to be a specific susceptibility to the chemical activity of light rays, or to a precocious senility of the skin (Forster). The iron-free pigment lies chiefly in the basal layers of the spinous cells, and is probably brought to them from the chromatophores with their outrunners, which are present in large numbers in the corium. Degenerative or collagenous changes, such as occur in elastic tissue, then produce white, scar-like, flat, atrophic areas, with or without stellate telangiectatic vessels, or perhaps horny warts

from hyperkeratoses of the stratum corneum, or actual cutaneous horns from collections of cornified material on the longer papillae. Furthermore, larger flat or mushroom-shaped angiomas occasionally appear with eventual transition into endothelial angio-sarcomas (Wesolowski); ulcerating carcinomas have arisen from the cutaneous warts (Kreibich, Kerl), etc. Councilman and Magrath believe these are analogous to many of the carcinomas of older people, and to x-ray carcinomas (p. 2084), in which the epithelial overgrowth is likewise dependent on, or related to, atrophic and other changes of the cutis. Moreover, they have another point in common with xerodermal carcinoma, viz., the absence of metastases. The sarcomas may be pigmented or not; in the former, probably the chromatophores of the skin manufacture the pigment taken up by the tumor cells (Wesolowski). Xeroderma pigmentosum is also frequent in the eyes and their neighborhood (Bayard).

For general considerations of pathological pigmentation, see Hueck in Handbuch v. Krehl-Marchang VI, Leipsig, 1921. [For curious hyperpigmented lines of the thorax and abdomen of a negro infant, see Weidman, F. D., J. Cut. Dis., 37: 517: 1919.—*Translator.*]

Leucopathia, or Leucoderma, or Pigment Atrophy

If the normal skin is poor in pigment the condition is known as leucopathia. It may be congenital (albino), or acquired (vitiligo). Generalized congenital leucopathia is called albinism universalis. The pigment is lacking in the rete as well as in the bulbs and the shafts of the hairs.

Albinos have a white, or rosy, transparent skin and yellow to flaxen white silky hair. The iris, the chorioid and the retina are also nonpigmented and transparent, (high degrees of photophobia and nystagmus). Universal albinism is frequent in the darker races. Heredity, and consanguinity of the parents are important factors in its origin (Seyfarth). According to Dalpe, the albinism does not extend in the bed of the nail to the ends of the finger, but commonly only half way (Rille). Partial congenital lack of pigment, albinism partialis, occurs in negroes (lit. and illustrations by Meirowsky), and in the white races, *e.g.*, in the hair (poliosis, leukotrichia circumscripta).

Vitiligo is the idiopathic form of acquired leucopathia; it is more frequent in the Negro than in the Caucasian race. In the early stages, white spots, immediately surrounded by a dark brown zone, appear in preferred situations. The hair is also bleached in the neighborhood. Then the rounded or oval spots increase in size, the pigment is pushed toward the peripheries, and surrounds the pale places with dark, concave edges. Later, the darker areas between the white spots are much more prominent by contrast, and the temptation is to regard the brown spots

as the pathological condition. The entire skin may be involved, and after many years, may appear bleached with the exception of a few dark stripes or islands.

Histologically, pigment is absent, or almost so, in the white areas, and increased in the rete cells and the hair in the other situations. Very few pigment-containing wandering cells can be found in the corium. The neighboring lymph nodes may be stained light brown. The etiology is unknown.

In the differentiation from leucoderma syphiliticum, it may be considered that the white spots of vitiligo contain isolated situations, wherein are fresh deposits of pigment, or remnants of the older pigment retained (Lang).

Local acquired leucopathia is frequent as the residium of various inflammatory and cicatrizing processes, thus, after the healing of furuncles, variola, lupus, leprosy, syphilis, and excoriations from pediculi.

Bleaching is due to the removal of pigment by wandering cells to other situations of the skin or to the lymph nodes; or the pigmented rete cells are destroyed and, in the atrophy of the corium and the rete, the pigment-producing and pigment-retaining structures disappear (Kaposi).

Leucoderma syphiliticum (Simon, Neisser), is of great practical importance. It presents itself as white spots surrounded by brown, varying from the size of lentils to pennies; these coalesce to arched figures, or networks, chiefly in situations normally more strongly pigmented. The condition is more frequent in women than in men. They are located in the nape of the neck, the neck itself (collar of Venus), or where the clothes are tightly applied (inguinal region). The discoloration is more easily recognized in brunettes. It is an involution phase of a macular or papular syphilid, but similar pictures are also produced by other causes, such as toxic damage to the skin from renal or neurogenic disturbances, (according to Freymann, the first named cause is not very frequent). It usually begins during the first year after infection. It heals quite slowly, sometimes after years, by the deposit of new pigment; during this time the varying lack of pigment is the only change in the areas. More rarely, it leaves isolated scars. Since the spots are easily differentiated by their localization, size and arrangement from leucopathias of other kinds (Lang), the condition is quite important in the diagnosis of previous syphilitic infection. Lit. by Brandweiner. For its relation to Block's dopa-reaction, see Gold and Reiss.

The rare, so-called, "white spot disease," in which chalky white, smooth spots appear as though imbedded in entirely normal skin, is dependent upon a pathological change of the connective tissue in the upper layers of the cutis (lit. by Riecke).

Acquired Hypertrophies of the Skin

By callus is meant a situation subjected to repeated or continuous pressure, (hands, feet), and covered by a circumscribed hypertrophy of the horny layer of the epidermis. The name tylosis, or keratodermia palmae et plantae, is used when the cornification is diffuse. It also occurs independent of pressure, thus congenitally (p. 2172), or because of neurotic or toxic conditions, *e.g.*, after the internal use of arsenic (Schutz). Dubreuilh (lit.) saw carcinoma develop on the base of an arsenic keratosis; according to Ullmann (lit.), this is quite frequent; even multiple primary growths have been observed. The inorganic arsenic preparations, such as sodium and potassium arsenate, or Fowler's solution, are the more important, possibly because they are retained for many years, (decades),

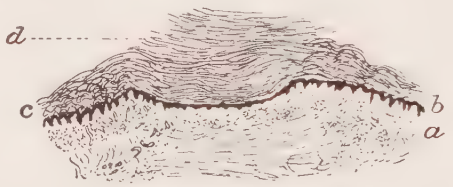


FIG. 1029.—Corn, half diagrammatic; a, corium; b, papillary body with rete Malpighii; c, hypertrophic cornified layer; d, stratified horny layer.

particularly in the liver, and are continuously excreted by the skin. Corns (clavus) are likewise hypertrophies of the horny layer, but are flat and the papillary bodies are squeezed together (fig. 1029).

Jadassohn classifies these callosities, such as the clavus and the arsenic keratoses, under the epitheliomas (p. 2196).

A cutaneous horn is a hypertrophic horny layer in the form of a simple bent, or a spirally wound, goat's horn-like structure, in length 30 cm. or more, and a number of centimeters in thickness. It appears solitary or multiple on the scalp and in the face, particularly on the nose, on the backs of the hands, on the tongue, and on the penis. The skin from which it arises may have been healthy or scarred (burns or eczemas), the site of clavi, warts, atheromas, carcinomas, etc. H. Burckhardt (lit.) believes it is an epithelioma (Jadassohn). They are more frequent in women than in men. (See p. 1538 for the structure of a cutaneous horn; lit. in appendix.) Concerning the process of cornification, see Martinotti.

The ordinary hard wart (verruca vulgaris), appearing by preference on the hands, (around the finger joints and around the nails), in both children and adults, is an epithelial overgrowth, combined, as a rule, with enlargement of the skin papillae. Verruca dura plana (juvenile), and verruca dura verruciformis s. papillaris are differentiated. The verruca vulgaris is acquired and transplantable (Jadassohn).

According to Unna, a wart begins histologically as a laminated thickening of the spinous and horny layers (fig. 1030). When fully developed it consists of an inner papillary stalk broader at the top, separating into a number of thready papillae, covered by epidermis, with thickened spinous and horny cell layers. The complicated papillary covering is not a product of active overgrowth of the corium papillae, but the result of "plowing up" of the corium by the growing epithelium. In later stages the wart is cleft; bag-shaped, horny complexes are separated from the interpapillar parts, whereupon the papillomatous structure becomes visible (fig. 1031). According to Unna, furthermore, the hard wart is a benign epithelial tumor of the upper skin, which he also calls akanthoma because the hyperplastic process involves the spinous layer; to this akanthosis is quickly added an hyperkeratosis. Unfortunately, the term "akanthoma" is also used to describe a cornifying carcinoma, (called by many dermatologists a spinous or a prickle-cell carcinoma), *e.g.*, by Ewing, a term which leads to confusion.

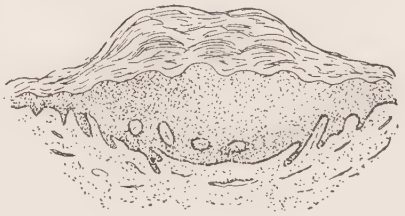


FIG. 1030.—Verruca dura plana (infectiosa); half diagrammatic; comp. text.

Condylomata acuminata occur particularly on the external genitals, and in the neighborhood of the anus, (on moist places, fig. 796, p. 1537), as cauliflower-like growths as large as a child's fist.

FIG. 1031.—Group of old warts showing their papillomatous character after detachment of upper horny layer; b, corium with glands; d, papillary structures projecting above the skin level a; c, subcutaneous and fatty tissue. (Magnifying glass.)

Carcinomata have arisen from them, but rarely; see the observation of the author on p. 1705.

The pointed condyloma consists microscopically of many branched papillae which increase in number on further growth of the structure; the stalk contains blood and lymph vessels, spindle cells and mast cells; the papillae are covered with luxuriantly growing, thickly layered, epithelium, the chief mass soft and uncornified, and not, as in a wart, hyper-

keratotic (fig. 796, p. 1537). The condyloma may be called a papillary fibroepithelioma on account of its histological character; according to Unna, it is an epithelial tumor, a pure akanthoma, beginning as a laminated thickening of the epithelium, and although its further growth is due partly to proliferation of connective tissue, the real active part is the epithelium producing new papillae from the growing stalk.

Acanthosis nigricans (Unna), or dystrophia papillaris et pigmentosa (Darier), is a rare disease, recently observed more frequently, consisting of multiple flat, brown black nodules, rough and dry in consistency. Various parts of the skin, particularly the abdomen, but also mucous membranes of the mouth and the nose, may be attacked. The brown pigment appears in the enormously overgrown spinous layer, and in the cutis, which also has collections of leucocytes and dilatation of blood vessels. The upper horny layers are markedly thickened to an ichthyosis-like condition of the surface which becomes like a file or like shagreen leather.

It is peculiar that acanthosis nigricans occurs relatively frequently in serious internal diseases, particularly malignant neoplasms of the abdominal organs (in thirteen cases collected by Spietschka, it occurred in nine women and four men; in five cases there was a gastric carcinoma, in three cases, uterine carcinoma, and in one a breast carcinoma; see lit. by Burmeister, Bogrow, Pribram). According to Darier's hypothesis, supported by Couland, the abdominal tumor, by its situation, involves the sympathetic nerves which produce the dermatosis (Janovsky).

Epithelioma molluscum (p. 2156) also belongs to the acanthomas.

Acquired **elephantiasis, pachydermia acquisita,** is an hyperplastic thickening of the cutis, subcutis, and perhaps also the epidermis, endemic in many tropical and subtropical regions; in Europe it is sporadic and usually from other causes.

The chief site of the disease is in the lower extremities, particularly the thighs, and in female (p. 1537) and male (p. 1703) genitalia. In severe cases, the circumference of the thigh, or the entire leg, is increased two to three times the normal; they change to plump, monstrous, bloomer-like cylinders which blend, without separation, on observation from in front into the pillow-like back of the foot (elephant's foot, fig. 1009). The intermuscular tissue is also infiltrated by the connective tissue overgrowth; the muscles, fat, and nerves are gradually destroyed. The bones are thickened in very irregular ways by periostitis ossificans. Osteomas may appear in the connective tissue of the cutis. See under the respective organs for elephantiasis of the penis, scrotum, female genitalia, etc.

The parts are hard, bacony, tough; the poorly cellular connective tissue is composed of many crossing fibrillar bundles; or they are soft and

gray white when composed of mucoid edematous tissue. The consistency is not uniform throughout, but may even show focal richly cellular granulation tissue. The surface of the skin is smooth (E. glabra) or papillary (E. papillaris or verrucosa) or nodular (E. tuberosa) from focal, hard hyperplasias of the cutis and subcutis. The horny layer may be folded and thick, as in ichthyosis. Occasionally, the skin is pigmented brown or black (E. fusca, nigra).

The surface may ulcerate and weep; the exuded fluid is often milky white. Lymphorrhages are common, particularly in the forms with varicose lymph vessels (E. lymphangitica s. lymphangiectatica), especially frequent in the genitalia, but also in other regions, *e.g.*, an entire leg.

FIG. 1032.—Lymphangiectatic crural elephantiasis, girl 19 years old; development gradual, non-inflammatory.

Etiology.—(*a*) It may be congenital, or related to a congenital or hereditary anlage, even though the growth of the part to its monstrous size occurs many years later (fig. 1032). In these cases there is tumor-like thickening of the skin, represented by part fibroma with lymphangiectasias or hemangiectasias, and part neurofibroma as discussed on p. 2026. (*b*) Endemic tropical elephantiasis arabum is produced by obstruction of the lymph vessels with the filaria sanguinis hominis (Lewis) (p. 210), which, in addition to lymph stasis, produces chronic inflammation of the skin and swelling of the lymph nodes, chiefly of both legs, and the male genitalia. (*c*) Other cases known as elephantiasis nostras, result from chronic and recurrent skin inflammations of an erysipelatous, lymphangitic or eczematous character, all producing circulatory disturbances, particularly of the lymph; there are first, swelling, and then hyperplasia of the tissues. The influence of lymph stasis is seen, *e.g.*, in elephantiasis vulvae or scroti after removal of the inguinal nodes, or of the arm after removal or infiltration of the axillary nodes in breast carcinoma, etc. (fig. 97, p. 176). Other, often enormous, overgrowths of the labia and clitoris appear spontaneously. End results of lupus sclerosis of the extremities (p. 2126), or superficial skin tuberculoses with involvement of the lymph

vessels (p. 175), or occasional syphilitic ulcers (fig. 1020) may be elephantiasis because of lymph stasis and subsequent connective tissue hyperplasia. The type following varices and varicose ulcers, with their additional inflammations, lymphangitis and lymph stasis, are called phlebectatic pachydermia, or elephantiasis phlebectatica (fig. 1009, p. 2112, and fig. 95, p. 169). Lit. in appendix.

Scleroderma is a rare skin disease of local or general extent, in which the skin swells and becomes board-like in its hardness, subsequent to hypertrophy and sclerosis of the connective tissue, all in a fairly short course of time (sclerema elevatum). The situations undergo later atrophy, become thin, flat, pigmented, parchment-like, stretched and too small (sclerema atrophicum). Since the shortened, atrophic skin is tightly adherent to the underlying muscles and bones, interference with joint functions to actual contractures, are common. Even respiration may be difficult. Symmetrical sclerodactylia often begins with the symptoms of Raynaud's disease. Atrophy of bones and even mutilations occur. The cause of scleroderma is unknown; it is believed, by many, to be a "trophoneurotic" affair as reflected in the name "angio-trophoneurosis" (Cassirer and Oppenheim); Hornowski considers it a vessel neurosis as in erythromegalia and Raynaud's disease (Schwimmer, lit. Luithlen); others saw the condition accompanied by disease of the thyroid gland, (Nobl, Braun). Herzog saw cystic degeneration of the spinal ganglia (Marburg) in a case of progressive scleroderma. [In a case of congenital scleroderma and sclerodactylia, Weidman found enteritis, meningitis and possible syphilis; see Arch. Dermat. and Syph., 1: 375: 1920.—*Translator.*]

Sclerema neonatorum is a disease of the newborn wherein the skin becomes hardened over the entire body, but without showing anatomical changes in structure. Langer holds that this condition is brought about by a change in the proportions of olein, palmitin and stearin, *i.e.*, of the glycerin esters of the fatty acids; the fat contains more of the palmitin and stearin, which have higher melting points—46° to 53° C.—than the fat of adults, which contains a greater amount of fatty acids, lipochromes and iodine, and melts at 45° C. After loss of fluid, and lowering of the temperature (in conditions of collapse), the almost colorless fat of infants, hardens because of its higher melting point. According to Knopfelmacher, the percent of olein which melts at ordinary temperatures in the skin, gradually and regularly increases during the suckling period; in the twelfth month it has reached almost the figure of that in adults (65%). Even in the second month of life, the olein content is usually so high that fat sclerema rarely develops. After six months, the development is impossible (Luithlen). See Tribuolet, Ribadeau-Dumas, Debre, con-

cerning fat "sclerema en plaques" with the accumulation of eosinophilic cells and a blood eosinophilia.

Hypertrophies from Congenital Anlage, Naevi, and Tumors of the Skin

Ichthyosis is a disease of the first years of life, hereditary, non-contagious, and caused chiefly by a growth anomaly of the epidermis, of mild or severe degree, producing thickenings of different appearances.

The skin of the rare cases of congenital ichthyosis appears as though covered by polygonal, plate-shaped, umbilicated, horny plaques, arranged

FIG. 1033.—Congenital or fetal ichthyosis in newborn; ⅓ nat. size.

FIG. 1034.—Piece of skin with ichthyotic thickening of epidermis; 9/10 nat. size.

in various directions with crossings, seams, and furrows; they cross around the joints, and are separated by thick layers of cornified epithelial cells enclosing woolly hairs. Horny layers also appear in the dilated hair follicles. The epidermis, six to ten millimeters thick, splits as in a tortoise-shell, where the body is stretched. Hebra compared the appearance of the brown red, map-like skin, glistening as though varnished, to that of the covering of a turtle, or a half-baked suckling pig; the term harlequin fetus is also descriptive. Because of the shortness of the tough skin, the fingers and toes are hindered in their growth (club hand, club foot); the skin reduplications around the eyelids and the lips disappear completely because of the tension; the tough mouth remains open from marked

ectropion of the lips (fish-mouth, diver's physiognomy). The ears are deformed, the nose is flattened, or completely defective (fig. 1033). Because of the plate-like or shell-like conformation of the horny plaques, the condition has also been called scutelatio. (Lit. by Riecke, Huebschmann, Fulci, Friboes.) It occurs chiefly in prematurely born infants which soon die. Milder forms in mature children, and compatible with life, are called ichthyosis congenita larvata. Etiologically, consanguinity of the parents is of importance.

In the ichthyosis of early childhood and lasting practically the entire life of the individual (keratosis), the skin is raw, dry, covered with scales, leaves, or thick plates of epidermis, or with horny warts. The masses are white, green or black, the furrows and lines of the skin are deepened.

Various degrees and forms are differentiated: in ichthyosis simplex, the extensor surfaces of the upper arm and the thigh are covered by pale red nodules about the size of the head of a pin, containing a scaly prominence in their middle, which, on removal, reveals a rolled up hair (lichen pilaris). More frequently, however, forms appear in which the skin is covered by dirty white, or gray plates of the size of lentils to nickels, sunken in their middles (ichthyosis scutelata), raised around their peripheries, shimmering, transparent, and producing a checkered appearance of the skin (ichthyosis nitida, ichthyosis nacree; mother-of-pearl-like). The true ichthyosis hystrix (hystricism, porcupine-men) is more rare, and shows horny and spinous pigmented protuberances as flat shields. Many cases show linear arrangements (Stowers, illustration). Often, conditions called ichthyosis hystrix, are, according to Unna, nothing but naevi lineares neuropathici cornei; see p. 2173. In porokeratosis, the cornification is localized chiefly to the excretory ducts of the sweat glands (Mibelli, lit.); this stubborn, chronic disease is outspokenly hereditary. Keratoma hereditarium palmare et plantare is a hereditary thickening of the horny layer in these situations (Vorner). Local, isolated ichthyosis may also be called ichthyotic warts; around the elongated papillae, the horny layers grow to large spheres. (Lit. concerning ichthyosis by Gassmann, Riecke.) See Martinotti concerning anomalies and disturbances of cornification.

Naevi and Tumors of the Skin

New Growths of the Connective Tissue Group.—Fibromas of the skin appear occasionally as solitary cutaneous and subcutaneous nodules with smooth or nodular surfaces. If small, composed of papillae, broader above, and covered by a horny layer, the term fibroma papillare is used (fig. 1035), but it is probably better to say warty or papillary fibro-epithelial tumors. Other fibromas are mushroom-shaped, or pear-shaped and

pedicled—fibroma pendulum (according to Lennhoff, better called cutis pendula)—and are particularly frequent along the back, the inner side of the thigh, and the labia majora (fig. 864); their consistency is soft or hard. More rarely they are multiple, and on section composed of interwoven bundles, or concentrically layered, occasionally prominent, small to larger soft fibromas (sometimes many pounds in weight), called, because of their softness, fibroma molluscum. In the differential diagnosis, cysticercosis of the skin must be considered (p. 2061); in this condition, however, the nodules are very hard. Multiple fibromas of the skin are usually neurofibromas (p. 2026); the plexiform fibroma was discussed in this chapter. A solitary fibroma molluscum may belong to the "forme frustes" of neurofibromatosis.

When diffuse neurofibroma is followed by elephantiasis and lobulated thickenings of the skin, the condition is known as elephantiasis neurofibromatosis, lobulated elephantiasis, E. mollis, or pachydermatocele (p. 2026).

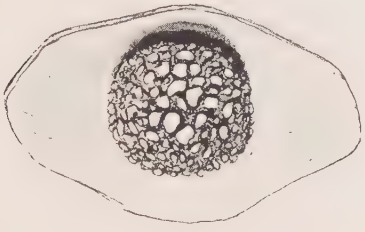


Fig. 1035.—Raspberry-like so-called fibroma (fibro-epithelioma) papillare from mons veneris; nat. size.

Fig. 1036.—Systemic papillary warty naevi (lineares) of skin of neck; woman 56 years old; factory worker; nat. size.

In the differentiation of E. neurofibromatosis and other forms of elephantiasis, we consider that simple fibrous elephantiasis, or simple skin hypertrophy, or pachydermia, is that condition in which the connective tissue parts of the skin and the subcutis undergo hypertrophy. The epidermis may be normal or thickened.

Neuropathic naevi, also called nerve naevi (Th. Simon), or neuropathic papillomas (Gerhardt), naevi unius lateris (v. Barensprung), or naevi lineares (Unna), or systematic naevi (Jadassohn), are papillary-warty, partly pigmented naevi, or merely ichthyotic naevus-like structures, arranged in lineal fashion, which, according to the earlier views, followed Voigt's border lines of the cutaneous nerve branchings (L. Philippson). Others have failed to find this relationship to nerve distribution (Hal-

lopeau), and Blaschko explains their origin and localization as growth disturbances on the boundary lines of a dermatoma. (Dermatomas are the isolated girdle-like segments corresponding to the primary segments, from which the skin was developed; see also Polland.) According to Jadassohn-Werner, most of the systemic naevi are localized in the direction taken by the hairs. Some of the systemic naevi originate from sebaceous glands or sweat glands (pp. 2197 and 2199).

FIG. 1037.—Scar keloid from neck of a young man; characteristic prolongations like crab claws.

A fibroma may undergo necrosis and calcification (Heising), occasionally leaving hard, irregular, multiple, subcutaneous nodules and masses up to the size of an apple in various situations, particularly the extensor surfaces of the hands, the forearm, the elbow, the sacrum, the nates, the knee, the fibula, the bones of the foot, (fibroma durum multiplex petrificans; lit. by Gergo).

The **keloid** is a smooth, flat, nodular, or irregularly wavy, branched, glisten-

FIG. 1038.—Cross section; nat. size.

ing, tough, elastic, pale rose, or tendonous white, sharply bounded, tumor. It appears (1) seemingly spontaneously, without an apparent cause, *i.e.*, from minor lesions, (scratches, Unna), and in this sense is regarded by many authors, *e.g.*, Wilms, Lexer, Braendle, as always of traumatic origin, while others esteem it a true keloid; or (2) it arises secondary to an obvious scar in those who probably have a congenital predisposition to its formation (for the histology of skin scars, see Levy). In the first case, the papillary bodies are at first unchanged, and the keloid is covered by normal epidermis; in its further development, however, the papillary bodies are smoothed out and flattened. In its beginning, the keloid is a connective tissue overgrowth in the corium, exceptionally rich in spindle cells, often arranged longitudinally along the vessels. The resemblance to a sarcoma, because of its cellular richness, has led to the opinion that keloid is really a cicatrizing fibrosarcoma. In its further

course, however, it shows an enormous overgrowth of, first, fibrous, then, homogeneous collagenous tissue, which determines the hardness of the keloid, whereas, in contrast to an ordinary cutaneous scar in which the first elastic fibers appear in two to three weeks and produce a rich, strong net-work, even after a number of months, no elastic fibers are produced (Schutz, Joseph, Wilms, Goldmann; Zurhelle also demonstrated the lack

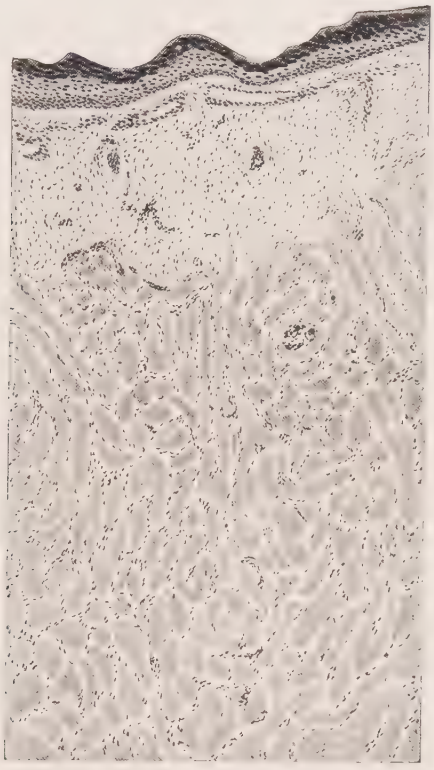


FIG. 1039.—Keloid from lobe of ear (due to puncture). The connective tissue of the corium contrasts sharply with the thick hyalin columnar fibre masses of the keloid; woman 24 years old. Van Gieson stain. (Medium power.)

of reticulum fibers). The author confirms these findings, as in the cases illustrated, and *e.g.*, in a keloid of the back of the foot following a lime burn in a twenty year old girl, and in a keloid in the scar of a burn over the sternum of a four year old boy. On the edge of the keloid the fibers of the unchanged subcutis were compromised by the growth, as though pushed aside, and the sweat glands were so separated that they appeared to be isolated parts of glands. By such means keloid tissue can be differentiated from physiological connective tissue, and from the connective tissue of fibromas and hypertrophic scars. The completed, typical keloid, therefore, is presented as a homogeneous mass of closely approxi-

mated, broad bundles of fibers, between which spindle-shaped cellular elements are present. The fibers may be arranged parallel to the surface and to the long axis of the tumor. According to Tomasczewski, the skin glands and the hair are always retained in true keloids; in a scar keloid, they may be absent. Others, for example, Heidingsfeld, can find no histological differences between the two. For further differentiation, hypertrophic scar tissue is usually quite tender, and is gradually dissipated in the neighborhood, whereas the painless keloid is always sharply demarcated from the cutis however far it spreads over the immediate region of the injury, although sometimes it is localized. Complete extirpation seldom removes the trouble for, in a few weeks, new ones are formed in the resulting scar. It never, however, produces metastases.

In negroes, because of a possible congenital predisposition (Plehn), keloids are often larger than fists in pierced ears, etc., or spontaneously. Others appear in scars of vaccinations, deep burns, irritations, ulcerations, wounds, surgical wounds, piercing of the ears, etc. True keloids select, by preference, the region of the sternum; according to Schramek (lit.), over 50% begin in this neighborhood, and are particularly frequent during the time of puberty and the years immediately following; females are more often the victims. They are often multiple, inasmuch as old sternum keloids are followed by similar growths on the breasts, the back, the upper arm and the abdomen. Occasionally an hereditary predisposition has been observed. According to Justus, there is some sort of relationship between hyperthyreosis and keloid formation. According to Payr, individuals with hypoplastic constitutions are predisposed to their formation, in fact, in general, to rich connective tissue formation, (adhesions). According to Menes, scrofula and syphilis predispose to keloids.

Myomas (leiomyomas) of the skin are rare, and, in contrast to the ordinary myoma, may appear in early years (hypothecated congenital anlage). They are solitary or multiple, small, warty nodules, seldom larger than a walnut. As points of origin, there have been considered the vascular musculature (Hess), as demonstrated by the author in a congenital tumor of the back as large as a dinner plate, or, particularly in multiple myomas, the arrectores pilorum (Jadassohn, Fritz, Simons and others), or the musculature of the sweat glands (Nobl, Gutmann and Sobotka, lit.). Rhabdomyomas are very rare (Schmorl).

Concerning leiomyomas, see also Dagonet, Kretzmer, Engel, Levit, Smilovici, Lieber (lit.), Zinsser.

Zieler, and Hayn (lit.), each describe a myoma sarcomatodes.

Lipomas of the subcutaneous tissue are frequent, always of lobulated structure, detectable externally; the lobules and lobes are held together by connective tissue septa. The isolated fat cells are large enough to be

seen by the naked eye. Lipomas may be differentiated from the subcutaneous fatty tissue by their lobulated and circumscribed structure, and also because of a differing structure and a lighter yellow to white color (fig. 1040).

FIG. 1040.—Lipoma on outer side of left thigh of an older, very obese, woman; cross section; ¾ nat. size.

Microscopically, there are fat cells (fat vesicles), of varying size, and in situations of more marked growth, indifferent pre-fat cells, which gradually contain more and more fat, at first finely granular, but later in

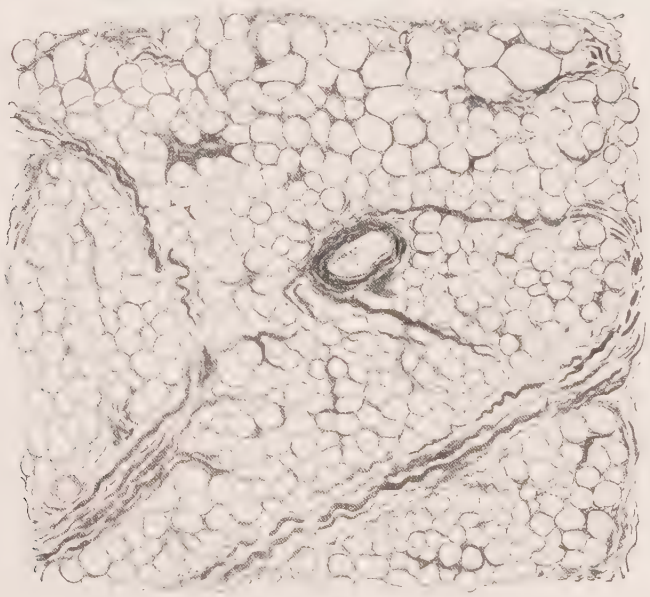


FIG. 1041.—Lipoma of subcutaneous tissue; connective tissue separates the masses of fat cells (with very varying size of cells) into lobules; vein in center in connective tissue. (Med. power.)

one large droplet; these cells, as in regenerative fat newgrowth, arise as endogenous cells (inside of older fat vesicles) as well as in the interstitial

spaces (Marchand, E. Rehn). Situations showing vigorous new growth are milk white and medullary (Rasor).

Lipomas of considerable size and weight, the growth of which becomes stationary at a certain time, mostly in old age, are particularly frequent around the shoulders and on the back, where they appear like broad or pear-shaped knapsacks; they also occur on the nates, the neck, the axilla, the abdominal walls, the thigh, etc. (Grosch and Stoll). Occasionally, a lipoma appears as a solitary, or multiple, pedunculated lipoma pendulum.

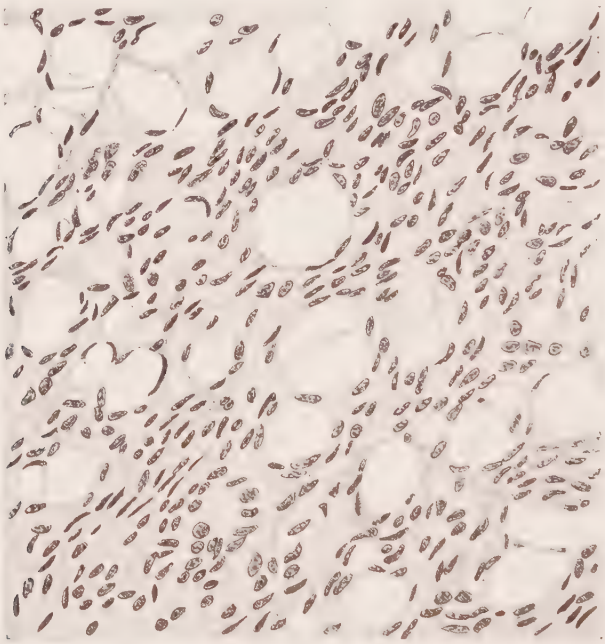


FIG. 1042.—Lipoma sarcomatodes. Skin tumor as large as a fist; cross section cloudy yellow white surface, growing quickly in skin, or under skin of r. lower leg of woman 31 years old. Many finely vacuolar, polymorphous fat granule-containing cells; larger drops between cells (no typical fat cells); medium power.

A lipoma in which the connective tissue is quite rich and tough is called a fibroma durum or a fibro-lipoma; one in which, in addition to fat tissue, there is sarcoma, or mucoid tissue, is called a liposarcoma (rare) or a lipomyxoma. A lipoma sarcomatodes, or also, lipoplastic sarcoma (Comolle, lit.), is a sarcoma, the cells of which tend to produce fat, *i.e.*, fat granules and larger droplets, but not actual fat cells; the grape-like connections of the cells are lacking (fig. 1042). Other lipomas soften in their centers; trauma may be the initiating cause. The fat cells become necrotic, are dissolved, and the result is a cavity filled with an oily or sebaceous mass (oil cysts); the wall may calcify. More rarely, a lipoma undergoes calcification in its entirety; this may occur after hemorrhage; otherwise

calcification takes place only in isolated necrotic situations of the lipoma. Ossification has also been seen. Thus, in an old woman with a lipoma as big as two fists in the right supraclavicular region, the author found an ossified part in its center almost as large as a hen's egg. Larger lipomas may ulcerate superficially and suppurate from external insults such as tearing or rubbing. Lipomas may furthermore be accompanied by important newgrowth and dilatation of vessels (lipoma telangiectodes). The skin over pedunculated lipomas may be pigmented brown (naevus lipomatodes), or may be covered by thick hair.

Lipomas do not participate in cachexia of the carriers, which illustrates their independence. According to E. Rehn, their fat is probably a permanent fat, in contrast to the wandering, or the usable fat, and thus is possibly the best transplantation material. At least, simple atrophy may take place when, *e.g.*, the nutrition is compromised by torsion of a pedicle; the skin over the lipoma then becomes folded. Lipomas are often present at birth, thus, in spina bifida; the same is true of multiple lipomas. Others are acquired later; pressure and rubbing are predisposing causes for their development. They are much more frequent in the female sex.

In the monstrous, diffuse, symmetrical lipoma formation in the necks of males between the fortieth and sixtieth years (Lenormant and Verdun, lit.), the subcutaneous tissue, and the subfacial and intermuscular tissue, are sites of formation (Madelung); accompanying diffuse lipomatous areas may also appear, chiefly in the subcutaneous tissues of the trunk and extremities. Many differentiate this condition from true lipomas, and call it circumscribed fat tissue hyperplasia in certain regions (lipomatosis regionaria, Billroth). Occasionally, a change in the thyroid gland is the causative factor (Turk). Symmetrical multiple lipomas are sometimes apparently related to the course of nerves and muscles. Thus the author saw multiple lipomas in both arms of a seventy year old, very adipose woman; the skin was irregularly nodular from numerous lipomas some of which were movable, and some as large as hen's eggs, the arms were irregular in contour, nodular, wavy, and markedly thickened; the regions above the wrist joints were suddenly demarcated from the hands by egg-sized nodules; the hands remained free of growths; the lipomata could be easily shelled out from the surrounding sacs in the subcutis and between the muscles, and on section were soft; one lipoma, as large as a cherry, was calcified. (Lit. by Payr and Meissner, Hasegawa.) Askanazy described multiple lipomata from a lipomatous change in lymph nodes.

Adiposis dolorosa (Dercum's disease), consists of isolated nodules, diffuse regional, or universal masses of fat, the latter cases resembling

adipositas universalis, but with more lobulation, and the masses are painful on pressure. The face and hands remain free. In addition, there are general bodily, and particularly muscle, weaknesses, nervous and psychical disturbances, and a tendency to general adiposity (lit. by Weiss, Schwenkebecher, Hirschfeld, (lit.), Klieneberger, Falta, Frowein). Some cases show changes in the thyroid and hypophysis, among them, tumors, thus carcinomas (lit. Strada). See Gunther for lit. concerning lipomatosis and its clinical forms.

Myxomas are rare, but occur in the cutaneous or subcutaneous tissues, or in the subfascial or intermuscular connective tissue; according to Ribbert, they are always originally myxomas. They are gray, mucoid, transparent, jelly-like or tougher, bacony, often with lobulated structure, and may reach considerable size. At times, they are present at birth.

Histologically, they are composed of a slimy, basic substance containing rounded, spindle, or stellate cells connected to each other by prolongations. Combinations occur with fibromas or lipomas, or marked fatty changes in the cells; transformations into richly cellular sarcomatous varieties called myxosarcomas, have also been observed.

Chondromas,—lobulated, cartilaginous tumors in the subcutaneous tissues,—and osteomas, are very rare.

For chondro-osteoma, see Carl, (lit.).

Older people are subject to cutaneous or subcutaneous skin stones consisting of calcium deposits, usually without ossification, in older inflammatory foci (granulation tissue), in the secretions of glands, in lymphothromboses, phleboliths, or tumors, *e.g.*, lipomas or lymphangiomas; but the calcium may be deposited in ordinary subcutaneous connective and fatty tissues (lit. by O. M. Chiari, Weissenbach, Kerl, lit.). Ossification occurs to mild degrees, *e.g.*, in the scalp (J. Schaffer); the process is tissue metaplasia (Orth), *i.e.*, the connective tissue cells are changed to bone cells, and the foundation substance to a bony substance, or cellular metaplasia produces osteoblasts from connective tissue cells and these form new ground substance. The calcium already present in the tissues, may initiate the formation of bone by irritation as in calcified dermoid cysts of the skin, or calcified epitheliomas (Sehrt, Strassberg, Murakami). Calcification in the skin also occurs as an expression of disturbance of calcium metabolism; too much calcium is present, or is not properly dissolved or suspended in the plasma. This condition may be called calcinosis, or calcium gout (Hollander, Drucker, lit., Pospelow, Liesegang). Very rarely, calcinosis is universal as a constitutional disease in which large amounts are deposited as strands or plaques in the subcutaneous tissues, the fascia, along the nerve sheaths, the muscle bundles, or the connective tissue septa; the internal organs, such as the lungs, the

stomach, the kidneys, otherwise favorite sites of calcium metastasis, are uninvolved (Dietschy, Tilp, Marchand, Verse, v. Gaza, lit.). Since the nodules are often deposited around joints, the condition is easily mistaken for ordinary gout. For calcium concretions in the subcutaneous tissue in progressive scleroderma or sclerodactylia, see Thibierge and Weissenbach, Oehme. See Jadassohn (lit.), for the rare psammomas of the skin in the posterior part of the scalp, particularly along the midline, similar to the intracranial dura psammomas; they appear even early in childhood. The hair falls out in these places, or merely a lanugo hair is developed. They may be regarded as developmental anomalies in the sense of naevi.

Hemangiomas of the skin, or angiomas in the narrower sense, are usually congenital, or, at least, appear during the growth period. At

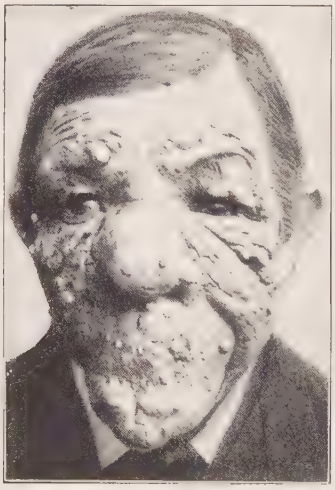


FIG. 1043.—Naevus vasculosus giganteus of face; the enormous upper lip below the thick nose.

times, they are birth marks (naevus vasculosus), of bright red or port wine color (naevus flammeus and naevus vinosus), occupying a small, or more extensive area, diffuse and flat, not sharply bounded, but little raised above the skin surface. They are in the upper layers or all the layers of the corium, the subcutaneous tissues, or deep in the fat tissues. They may be multiple in a certain region, and separate in the various layers of the skin.

Many hemangiomas lead to warty protuberances; others, with fibrous hyperplasia in the neighborhood of the blood spaces, produce nodular, rounded, lobulated elephantic thickenings (elephantiasis hemangiomatosa). Seifert believes the case illustrated in fig. 1043 belongs in this category (naevus mollusciformis elephantiasticus). But others are enormous, nodular, lobulated and purely vascular, and Riecke believes

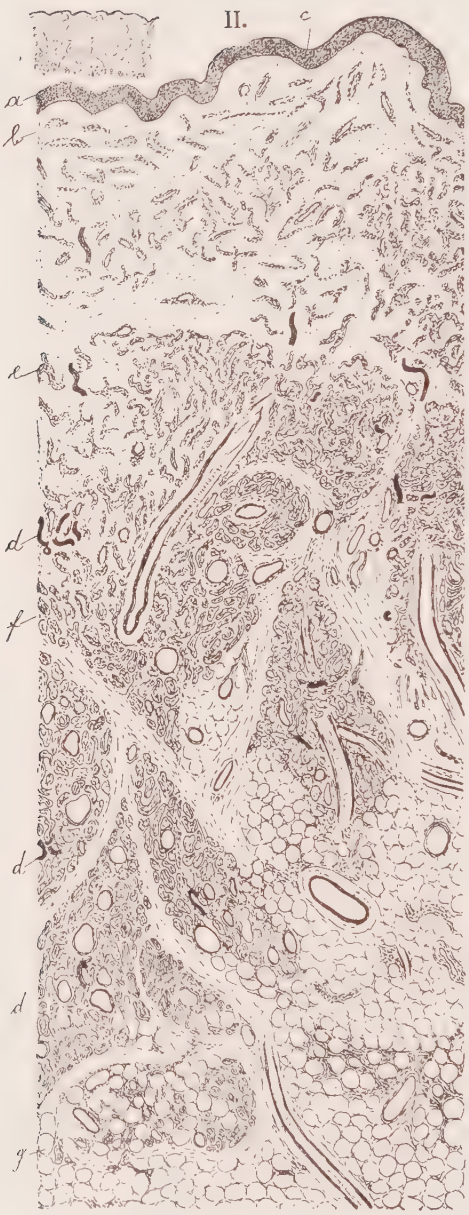


FIG. 1044.—From a congenital hyperplastic hemangioma (telangiectatic naevus) of skin of abdomen and chest of a nine months old girl; tumor the size of a dessert plate. I, natural thickness of tumor; II, microscop. sect.; a, epidermis; b, cutis showing hyperplastic capillaries; c, artery; d, parts of sweat glands; e, hyperplastic capillaries at border of cutis which form nodules and lobules in subcutis (f) and in subcutaneous fatty tissue (g). (Løw power.)

(fig. 1043), the histological examination proved this case to be such a naevus vasculosus giganteus. Stiefel described a hemangioma simplex of the neck which, being the size of a child's head, produced obstruction during childbirth.

Histologically, we may classify according to the formation of new vessels, or according to the dilatation of vessels: (1) hemangioma simplex, or telangiectasia, or plexiform hemangiomas, consist of convoluted, wide, thin-walled, capillaries with sparse veins and arteries. Some of the naevi vasculosi belong to this type. A particular form of telangiectasia is called hemangioma simplex hyperplasticum.

Histologically, the convolutions of hyperplastic capillaries (figs. 1044 and 1046), have arteries with nucleated and richly muscular walls on one side (1045-b), and veins on the other side. The capillary endothelial cells are richer in protoplasm than ordinarily (like epithelium), and are often arranged in a number of layers around the lumena. Occasionally, the capillary walls are richer in spindle cells, and thus are similar to arterioles, the rule in angiomas which remain fairly stationary. The hyperplastic capillaries form groups, nodules and lobules (fig. 1044), particularly dense in the border of the subcutis, and separated by connective tissue stripes, or, in the deeper layers, also by fatty tissue. Between them are larger vessel lumena. The masses of vessels, in thick sections, look somewhat like carcinomata, or like sweat gland complexes; in fact, larger,

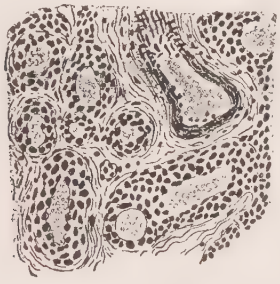
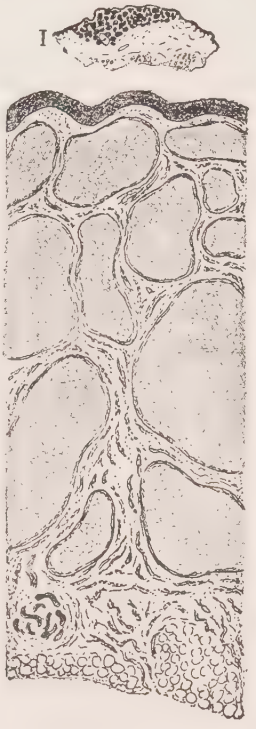


FIG. 1045.—Area from same prep. under high power; a, hyperplastic capillaries with several layers of endothelium, rich in protoplasm; b, small gartery; muscularis dark.



FIGS. 1046 and 1047.—Cavernous hemangioma of skin; I, macroscopical cross section in nat. size; II, portion of this under low power; a, epidermis; b, cavernous spaces; c, separating walls with hyperplastic vessels; d, sweat glands; e, subcutan. fatty tissue.

darker, arched pieces of sweat glands may be enclosed by them (fig. 1044-d). The capillaries also spread from the thicker nodules and lobules into the cutis and the subcutaneous tissues. The development of newer capillaries from budding of the walls of older capillaries is readily followed, particularly in the fatty tissue layers; the buds are solid at first and of equal thickness, the endothelial cells taking a prominent part in the process; later they become hollow tubes and establish communications with other vessels.

Hemangioma hyperplasticum is a congenital, flat, soft tumor which swells under pressure, abdominal compression, etc., of steady progressive growth, leading to thickening and mild waviness of the skin.

Particularly aggressive tumors stop at no tissue and have been found in the salivary glands (p. 599), (Konjetzny). See Schmitt, concerning deep angiomas of infiltrating growth in the orbit and the paratracheal regions.

Some look like soft warts. When the hyperplastic capillaries consist of practically nothing but solid cell strands, the name endothelioma hemangiomatosum may be used. Hemangio-endothelioma tuberosum multiplex (Jarisch), are multiple nodular tumors the size of millet seeds to lentils, appearing in the first years of childhood or even at birth, microscopically composed of tubular or cystic structures, showing transitions to capillaries (Oestreich and Saalfeld). Some believe they come from lymph vessels (lymphangioendothelioma tuberosum multiplex) or from sweat glands (syringo-cyst adenomas), sebaceous glands, or hair follicles. Elschnig and Guth (lit.) agree with Jarisch's interpretation. Regional multiple, subcutaneous, hyperplastic hemangiomas in the subcutaneous and fatty tissues produce movable, rounded, or arched, soft, elastic nodules, the size of millet or cherry seeds; on section they appear homogeneous, pale gray red, glassy, and show great similarity to small lymph nodes. When numerous cutaneous hemangiomas are also present, as in the described case, the deeper nodules might be mistaken for metastases. From excessive overgrowth of the vessel walls, an angioma becomes an angiosarcoma. When the interstitial tissue is markedly thickened, the tumor becomes a hemangio-fibroma.

See p. 245 concerning hemangiomatosis in which the cutaneous tumors are accompanied by similar structures in the spleen and liver; see Konjetzny concerning participation by the lungs, the brain and the muscles; see Borrmann for hemangiomas in lymph nodes, nerves and vessel walls.

Hemangioma cavernosum (tumor cavernosus) is less common than the telangiectatic form. It is composed of systems of many-shaped blue blood spaces separated by septa. Inside of the septa are numerous non-dilated vessels. In some cases, the tumor probably develops from a telangiectatic structure (fig. 1046); in others, the cavernous type is primary.

Occasionally, thrombosis and phleboliths (Fabian) appear within the vessels. More rarely, there is ossification in the hyalinized fibrous framework of a cavernous angioma (Glaser).

The site of cavernous angiomas is in the corium, where they may extend over larger areas, and reach below into the subcutaneous fatty layer; frequently, they grow exclusively in the latter; they may also penetrate as far as the intermuscular tissues. Prominent dark blue or red points, and

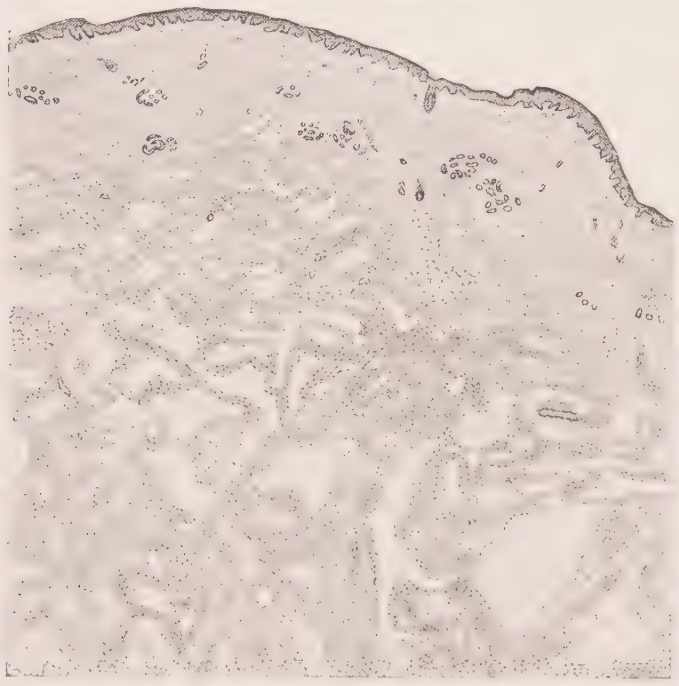


FIG. 1048.—Lymphangioma of skin of chest of a child 1½ years old; development in subcutaneous tissue; between the more dilated lymph vessel spaces numerous islands of fatty tissue; sweat glands in cutis. (Low power.)

flat nodules produce unevenness of the surface of the skin. Hemorrhages are uncommon even from thin areas.

A superficial situation of small cavernous blood vessels in the papillary bodies, as illustrated in fig. 1049, is rare. Very often, however, there are both blood and lymph vessel ectasias (hemato-lymphangioma), or still more frequently, blood from the capillary vessels appears in some of the spaces of a cavernous lymphangioma. Combinations with other neoplasms are fairly frequent, thus, with lipomas and fibromas. Many angiomas develop in situations in which there are fissures during the embryonal period. Such fissure angiomas, localized around the fissures or clefts of the face, are, at times, superficial, at times, deeper in the soft parts; their points of origin are probably displaced vessel anlage. Many angiomas begin as

small red spots distributed along the cutaneous nerve distributions. Those neuropathic angiomas (v. Barensprung, Simon), *e.g.*, of the face, may appear as naevi telangiectaticus along the distribution of the first and second branches of the trigeminal nerve. An angiokeratoma (Mibelli) is a superficial cavernous angioma with thickened horny layers. (Lit. by Guszeran.)

The question is still open of whether the frequent, numerous, small, senile, angiomas of the skin are actual new growths or "capillary varices" (Raff, lit., W. Pick, lit.). That they are found particularly often in carcinomas, as was stated (Leser), has been proved a mistake in statistics (Reitzenstein).

See p. 151; see lit. and critique by Schwarz, concerning the plexiform angioma.

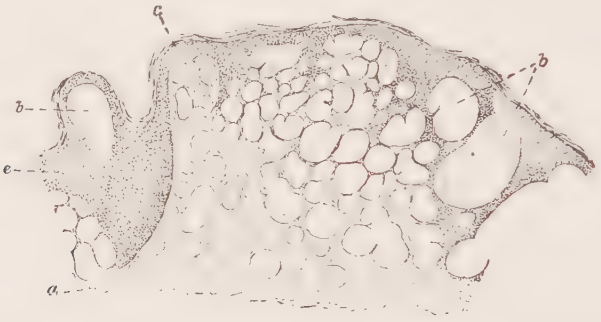


FIG. 1049.—Lymphangioma of papillary body of skin; varicose and cavernous vessels, (*e.g.*, at b) force themselves into the epidermis (e); vessels contain lymph or blood; some are empty; a, corium; c, papilla. (Medium power.)

The clinician considers telangiectasias as finely calibred vessels visible through the skin, and as Unna emphasizes, not capillaries, but veins in the papillary and subpapillary layers; they are seen, among other places, in cicatrizing atrophic processes, after x-ray damage, in chronic passive congestion and other conditions. For more details, see Miescher.

Lymph Vessel Tumors. Sharp differentiation must be made, if possible, between lymphangiectasias, which are simple dilatations of lymph vessels, and lymphangiomas, which are new growths. Lymphangiomas of the skin appear in many forms. They may be recognized as subcutaneous and cutaneous. The former are flat nodular thickenings, often diffuse, occasionally leading to elephantic thickening of the involved parts (fig. 1032).

On section, they seem to be dilated spaces lined by endothelium and filled with a clear or slightly cloudy lymph (lymphangioma cavernosum and cysticum). Their walls are fibrous and often quite muscular. The interstitial tissue contains fat and foci of lymphoid structures. The

consistency is usually soft, and after section of the tumor, the cavities collapse. When the connective tissue overgrowth is greater between the spaces, the tumors naturally are harder to the touch (lymphangio-fibroma).

Cutaneous lymphangiomas in the papillary bodies, with their rounded varicose and cavernous spaces lined by flat endothelial cells, may invade the epidermis (fig. 1049). Lymph sometimes seeps through the epidermis and produces vesicles on the surface, especially when inflammation is present. When wide blood capillaries are present and isolated lymph spaces are filled with blood secondarily, the name hematolymphangioma is used (p. 2185). Pigmented nests of cells may appear in the interstitial tissue.

Albrecht believes many lymphangiectasias are in reality malformations of the lymph vessels (lymph naevus). In this connection, Paltauf

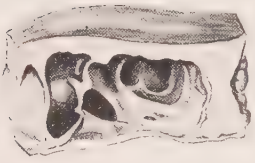


FIG. 1050.—Cavernous lymphangioma of skin of shoulder; cross section; nat. size; from a child.

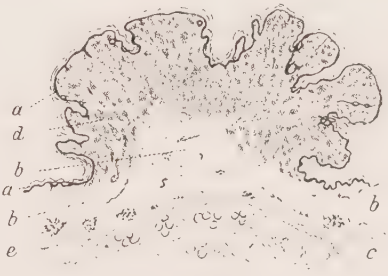


FIG. 1051.—Cross section of a so-called soft nodular wart (cellular naevus). The dark nests (d) consist of naevus cells; connective tissue in between; a, epidermis; b, cutis; c, subcutaneous and fatty tissue; e, sweat glands. (Magnifying glass.)

described an extraordinary lymphangioma cavernosum of the left hand and the left forearm, of progressive, malignant character, with extensive destruction of the bone, probably originating in a malformation of the lymph apparatus.

In other lymphangiomas, the endothelial cells are so overgrown that the name hyperplastic lymphangioma, or endothelioma lymphangiomatosum must be used. To this type of lymphangiomas belong the structures formerly called naevi pigmentosi, the lentigines and the ephelides (p. 2159), xanthomas (p. 2160), soft naevi (soft warts, fleshy warts, etc.).

The corium of these small tumor-like, though as a rule, stationary structures, contains strands, masses or foci of larger rounded, cuboid, or irregularly shaped cells, the origin of which v. Recklinghausen and others believe to be lymph vessel endothelium (fig. 1051).

Congenital warts, skin tumors, and in general, all diffuse or circumscribed skin anomalies, (Meirowsky), depending upon an abnormal germinal anlage for their origin, may be called naevi materni, or birth

marks, and according to their form, verrucose, tuberous, or papillary. Histologically, they vary considerably (cellular naevi, vascular or glandular naevi).

An older theory of Demieville, Lowenbach and others, stated that the naevus cells came from blood vessel endothelium or perithelium; later, Polland also hypothecated a relationship between naevus cells and the cells in the walls of blood and lymph vessels. Unna considers them embryonal epithelial cells, and his view has gained many adherents (Delblanco, Hodara, Kromayer, Thailhelfer, Scheuber, Waelsch, Marchand, Abesser, Judalewitsch, Kreibich, Kyrle, Bloch and Ryhiner, Miescher, Steden lit., and others). Naturally, malignant tumors which arise from these cell nests must then be called naevo-carcinomas. Schutz, Johnston, on the other hand, have adopted the endothelial theory; Riecke (lit.), in agreement with Riehl, believes in a connective tissue origin; according to this, the cells are derivatives of embryonal connective tissue cells, which undergo changes in form, and become pigmented secondarily as they increase in age. Ribbert believes naevus cells are a particular kind of connective tissue cells, *i.e.*, poorly differentiated "chromatophores" without pigment; Riecke believes that this hypothesis has not been substantiated and is of no value in the explanation of unpigmented naevi; the pigment, to which Fick calls attention, is secondary, and the pigment cells are not naevus cells, but rather the final pigmentation of naevus cells is proportional to the number of melanoblasts present (chromatophores). The author is of the opinion that naevi may come from both endothelial and epithelial origins; this idea, at least, explains the contradictory findings, and has been adopted by Frederic (lit.), M. Joseph, Ehrmann, Herxheimer and Bornemann and others. On the other hand, dalla Favera has recently agreed to an exclusive epidermal origin, and Fick believes the naevus cells as well as some of the branched pigment cells in the corium, which, he states, may undergo transition to pigmented naevus cells, are epithelial (see contrariwise, Ribbert, Kerl). According to Frieboes, naevus cells, as so-called "covering cells," arise from an organ composed of two germinal layers, but are not, however, true epithelial cells in the strict sense of the word, but are identical with the mother epithelial cells of mesodermal origin, *i.e.*, they are undeveloped physiologically, and come from the cells of the covering epithelium.

The boundaries of the cell nests are frequently obliterated in isolated situations.

In many cases staining for elastic fibers, or the use of Mallory's stain, has shown fibrous bands, and very fine fibrils surrounding the cell masses in the deeper layers, or even the individual cells, which speaks against an epithelial character of the growth (Kyrle later agrees to an epithelial

genesis, see p. 2188); also, cornification, protoplasmic fibrils, spinous and prickle cells are never present.

The soft, richly cellular, naevi (soft warts) are flat on their surfaces, or mildly irregular; they have been called lymphangiofibromas by v. Recklinghausen, but may simply be called richly cellular naevi; their cells are in networks, or more or less diffuse infiltrations of the connective tissue. When, however, cell nests invade the papillary bodies, the tumors are markedly nodular and papillary (fig. 1051). When the horny layer is thickened, the surface of a warty naevus grows harder (naevus varrucosus durus). The cell inclusions in the small papillae become covered by many horny layers, and practically disappear in mass in comparison to the amount of covering epithelium on the verrucose prominences.

See Martinotti for comprehensive lit. concerning endothelial tumors of the skin.

In pigment naevi, the nests are often particularly large, and the cells diffusely stained; some, however, contain yellow and brown granules.

Heavily pigmented spindle-shaped cells with prolongations, (chromatophores), are frequent in the connective tissue between the larger nests of rounded angular naevus cells.

Pigment naevi may be quite prominent (naevus pigmentosus prominens), nodular, or papillary. Other, soft, non-pigmented, warty naevi show abundant growth of hair (naevus pilosus). Lentigines (p. 2159) show histological pictures similar to soft warts; ephelides, on the other hand, do not (p. 2159).

Concerning the pigment (melanin)-containing cells, or forming cells, the views and the nomenclature are much confused. In the first place, there should be differentiated: (1) chromato-, or melanoblasts, *i. e.*, pigment formers, chiefly cells of the epidermis, and (2) chromatophores, pigment carriers, cells of the cutis, secondarily filled with excreted (dissolved) pigment, and probably of connective tissue origin (Tanaka). Bloch, and Bloch and Ryhiner, Oberndorfer, Bloch, and particularly his pupil, Miescher (lit.), believe that the failure of the "dopa" reaction, which is positive in the melanoblasts, and negative in the chromatophores, is a good argument in favor of this differentiation. ("Dopa" is an abbreviated name for dioxyphenylalanin, a substance regarded as a precursor of the pigment occurring in the skin, and which, in the skin, becomes changed to the granular, brown pigment melanin through the activity of an oxidizing ferment, the dopa oxidase.) Ribbert, on the other hand, is firmly of the opinion that the chromatophores (in his terminology), are peculiarly differentiated pigment-forming cells which lie in connective tissue, and in reexaminations, Heudorfer found that the dopa reaction is neither a specific fermentative, nor even an ordinary fermentative process,

but that a positive dopa reaction depends upon oxidation of dioxyphenylalanin by a primary reducing quality in the pigment and its precursors; thus the importance of the dopa reaction is again questionable in determining naevus cells and melanomas (Lemmel, Lignac).

According to Tieche, the blue naevi (benign melanomas, "chromatophoromas") of the skin, which have pigment cells in the fibromatous cutis, should be considered entirely apart from ordinary naevi (Ribbert).

See p. 2173 concerning systemic naevi, naevi lineares, naevi unius lateris.

FIG. 1052.—Fascicular fibrosarcoma of skin of back from woman 55 years old. Long spindle cells, and between this fibrous ground substance; it took 14 years for tumor to develop to present size. Recurrence after 8 years at same place (nodule the size of a hen's egg). Death from chloroform narcosis; no metastases found at post mortem; 7/8 nat. size.

The adenoma sebaceum and sudoriparum (p. 2197) are also classified with the naevi (organ naevi).

The xanthoma (p. 2160), according to Touton, is an endothelioma lipomatodes.

These various pigmented and non-pigmented naevi are of considerable practical importance because malignant tumors of a pigmented or a non-pigmented sarcomatous nature, but, according to others, also carcinoma, (according to the author, both), may arise from them. [For the melanomata in general, consult the comprehensive monograph of Dawson, J. W., Edinburgh Med. J., 32: 501: 1925.—*Translator.*]

Sarcomas of the skin may be present at birth, or appear in normal situations of the cutis and subcutis; when trauma initiates the tumor, it is probably because of some sort of added constitutional anlage; most frequently they arise in an already existing naevus. (For statistics, see

Lowenstein, B. z. k. Ch. 48, 1906; de Quervain following a wound from a splinter of a hand grenade, Med. Ges. Basel 17, II, 1916 and Eunike, D. Z. Chir. 151, 1919. See also Beck, M. 1922, 17, zur Frage des Rontgensarcoms.) More rarely they originate in a cutaneous ulcer. Various forms are recognized of primary sarcoma of the skin appearing in various sizes, usually roughly nodular, or pedunculated and fungoid. They are fairly tough, and composed of large or small spindle cells; the longer the cells, the tougher the consistency. The section surface is gray red or gray white, glassy and fibrillar; when the section surface is broken apart, the cell fasicles may often be seen macroscopically. Many spindle cell sarcomas are tough fibro-sarcomas (fig. 1052), others are myxomatous in

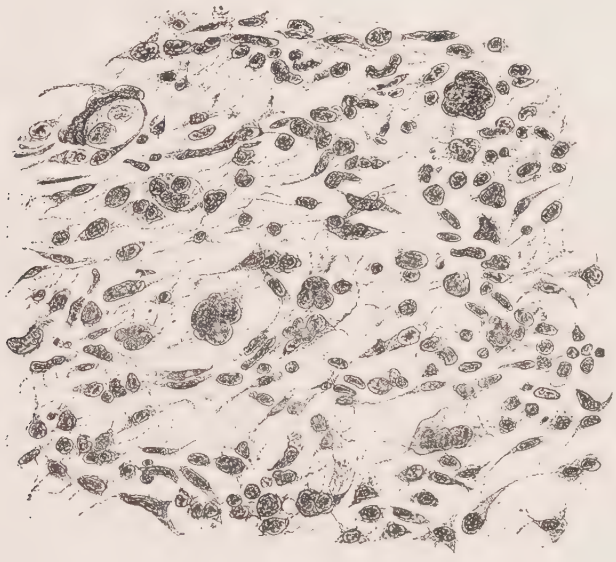


FIG. 1053.—Myxosarcoma, marked polymorphy (giant nuclei), from hand of child 1½ years old. (High power.)

parts; real myxo-sarcomas have often been seen by the author even in very small children. Giant cells are occasionally a striking part of the picture in a mixed-cell polymorphocellular tumor. Others are round cell sarcomas, of soft consistency with larger or smaller cells; the former are frequently alveolar in arrangement, inasmuch as cellular fibrous bands pass through the tumor tissue (p. 1210). Still others are malignant lympho-endotheliomas, coming from the lymph vessels (lit. Nather). Hemangio-sarcomas are also fairly frequent.

Among the latter are relatively tough forms, in which, as in a beet-like tumor of the back of the hand, there were thick cloaks of spindle cells around relatively narrow vessels (figs. 632, 633, pp. 1211, 1212). The spindle cells of this cylinder with a vessel in the long axis, are often arranged

in whorls because of their various directions of growth; the cell masses disappear in many situations, and the angio-sarcomatous character changes into that of simple dense spindle cell sarcoma. Other angio-sarcomas are softer and bloody; in one as large as a fist, observed by the author in the skin of a man's back, there were wide capillary blood vessels surrounded by exquisitely radiating spindle cells, or large round cells, or cells of a more cylindrical character (perithelioma). Whereas, in the above named forms of angio-sarcomas, the variations of which might be extended, the cell growth proceeds particularly from the adventitia of the vessels (perivascular sarcomas), there are other less common tumors, in which the endothelium of the vessels is the point of origin (endothelioma; Haslund). Alveolar sarcomas are not always easy to differentiate from carcinomas, unless the alveolar structure is lost here and there, and the simple sarcoma structure is brought to light; see pp. 1210, 1211. In the differentiation of doubtful cases, it is recommended that methods be used such as were described by Kuru, *i.e.*, the staining of the reticulum fibers by the method of Bielschowski, which was designed to demonstrate nerve fibrillae; by this method, the prolongations of the connective tissue fibers are made visible to much finer degrees than with the van Gieson method; these fibers separate the isolated cells of the sarcoma, while in carcinoma, the cellular masses hang together and are surrounded by a wide-meshed alveolar connective tissue structure (stroma); Romano, however, believes that this method is not diagnostically important. More work is necessary to decide this point.

Other sarcomas are telangiectatic, and show tendencies to hemorrhage. The tumor consists of innumerable capillaries or cavernous blood spaces, widely dilated, between which the sarcoma cell masses are embedded; the latter take their origin from the islands lying between the capillaries, and not as in angio-sarcomas from growth of parts of vessel walls or their adventitiae. Such tumors usually have iron-containing hematogenous pigments of yellow or red color. Telangiectatic and angio-sarcomas are occasionally combined.

The sarcomas which arise from naevi, (the more frequent), come from both unpigmented and pigmented, so-called fleshy warts, as well as from simple flat pigment naevi, about which details were given above (pp. 2187 and 2188).

The dispute concerning the correct nomenclature for malignant melanotic tumors (melanomas) depends upon (1) the question of pigment formation (p. 2189), and (2) the question of the nature of naevus cells. No unanimity has been reached. The nature of melanomas has not as yet been definitely established; Ewing (lit.), agrees with this statement. Ribbert, who upholds their connective tissue origin, *i.e.*, pigment forming

cells called, by him, chromatophores, calls these melanotic tumors, chromatophoromas. Kreibich expressed the opinion that the melanoblasts come from the epidermis into the cutis, and become either naevus cells, or the tumor cells of melanoblastomas; he, therefore, speaks of melanocarcinomas; he probably goes too far, however, when he considers that all melanotic tumors are epithelial, and denies the possibility of the origin of any mesodermal pigment-forming tumors (see Martinotti). It is probably better at present to retain the division into epithelial (melanotic carcinomas), and connective tissue (melanotic sarcomas) tumors. Both kinds can perhaps be grouped under the neutral designation of melanocytoblastomas (Lubarsch).

Melanotic tumors may, however, appear as primary growths in certain favored situations of the skin. These sarcomas are uncolored, or colored

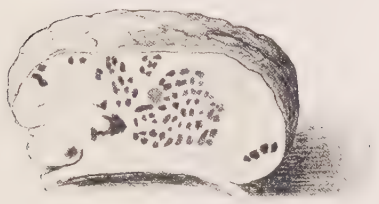


FIG. 1054.—Melanosarcoma from back of foot of woman 48 years old; 7/8 nat. size.

FIG. 1055.—Pedunculated melanosarcoma from back of woman 51 years old (histologically alveolar); 7/8 nat. size.

alveolar tumors, the pigment being autochthonous from the cells (the nuclei are not stained), or from chromatophores (p. 2189); round cell (often large cell or alveolar), as well as fascicular spindle cell tumors, also occur.

The alveolar sarcomas are the most frequent; these cells are rounded or rounded angular, epithelial-like, gathered into alveoli, some free of pigment, others densely pigmented, some diffusely so, and often necrotic over large areas. The stroma contains pigment, perhaps in large amount, contained in spindle cells and rounded wandering cells, so that cell contours and nuclei are hidden. In these cases, all the evidence is against melanoblasts (these are the cells joined in alveoli), and chromatophores (these are the cells which merely take up the pigment and do not prepare it). In contrast to these, there occur still other sarcomas composed of nothing but pigment cells of spindle shape, often arranged in networks or fasciculi, which must be considered as pigment producers. To this class, also, belong undoubted fascicular spindle cell sarcomas, as well as the practically important, quite poorly cellular tumors in which the pigment cells form a system of branched structures between connective tissue

fasciculi, the pigment appearing as though injected; the author has seen many tumors of this type which ran a clinically benign course.

Sarcomas also arise from naevus vasculosus; these are the above mentioned angio-sarcomas.

A number of cases of this kind examined by the author, did not give a clinical malignant impression because of their very slow growth. But wildly malignant pigment sarcomas occasionally appear in combinations of naevus vasculosus and pigmentosus.

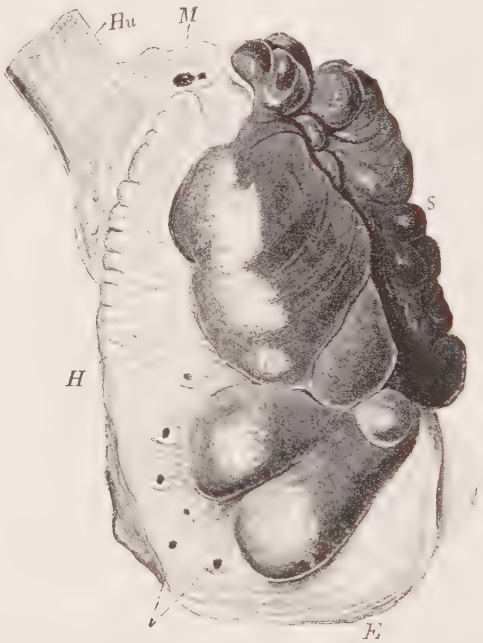


FIG. 1056.—Melanosarcoma, lobular, nodular, as large as a fist S., of the skin (H) of left elbow area (E); surrounded by disseminated metastatic tumors (M); Hu, humerus. Servant girl 44 years old; metastases in lungs, liver, brain, bone, etc.; $\frac{3}{5}$ nat. size.

The pigmented brown or black melanosarcomas (fig. 1056) are characterized by extraordinary malignancy; not infrequently they undergo marked softening. By regional and general metastasis through the blood and lymph channels, both pigmented and non-pigmented or checkered nodules appear in many organs and lead to death, even in a few months.

But the author has seen some which grew slowly, *e.g.*, a melanosarcoma as large as a hen's egg, in the vascular sheaths of the thigh, which appeared as a recurrent growth in two years, and then merely infiltrated the overlying skin (63 year old man).

The skin, all internal organs, and the entire skeleton, may be invaded by tumor nodules, some pigmented and others not. The liver, in particular, is full of tumors.

In certain, uncommon, cases of general melanosarcomatosis, diffuse melanosis of the skin appears as a brown to black pigmentation in regions free of tumor nodules; general melanosis also occurs in all organs in which the connective tissue cells, vascular endothelium, reticular cells and kidney epithelial cells are preferred (M. B. Schmidt, Matsunaga); in fact, only the nervous system may be free of the pigment. The skin itself shows the pigment, preferably in its connective tissue cells. It comes from disintegrated sarcoma cells, is carried about in a dissolved, reduced, nonpigmented form, and then is oxidized to brown, granular, intracellular deposits in the cells; it may also appear in the blood serum, in transudates, or in the urine. But melanuria (melanogenuria) may also be present without melanosarcomatosis (Feigl and Querner, lit.); the freshly voided urine is of normal color, but on standing, an increase in acidity leads to gray or black discoloration. If fresh urine is treated with iron chloride or chromic acid, the melanogen is changed into black clouds of melanin. (Lit. concerning melanin by Oberndorfer, Treuherz, Kutschera-Aichbergen.) Occasionally a urine is black when voided (Feigl and Querner). [For malignant melanotic tumors in the negro (rare), see Bauer, J. T., Bull. Ayer Clin. Lab. Pennsylvania Hosp., Philadelphia., December, 1926, page 5.— *Translator*.]

Metastatic skin sarcomatosis may appear from bone or intestinal sarcomas. Schlossmann (lit.) described a case of general sarcomatosis, particularly of the skin, in which the doubtful primary tumor was in the pancreas of a newborn child.

Sarcoma idiopathicum multiplex hemorrhagicum pigmentosa should probably be put in a separate classification; it is also called sarcoid (Kaposi), since it occupies a position between granulomas and sarcomas (de Amicis). But its structure of round and spindle cells around vessels is not that of a sarcoma (Selhorst and Polano and also Ehrmann). It is said to be favorably influenced by the administration of arsenic. Other forms of primary skin sarcomatosis may show a localized growth (Joseph), or even undergo regression (Spiegler, lit., Polland). This fact probably also requires that they be put into a separate class (Pini, Polland, lit.); C. Sternberg, who believes that the spindle cells are smooth muscle fibers, speaks of hamartomas, *i.e.*, a combination of lymph- and hemangiectasias with an overgrowth of smooth muscle fibers, but he leaves the question open if this is true of all cases. According to Friboes (lit.), this sarcomatosis cutis is not a disease per se, but appears in clinically and histologically different sorts of diseases. Friboes describes primary small cell lymphocell-sarcomas composed of connective tissue, which he calls lymphosarcomas. In a case of Krystalowicz, the tumors had a mixed round and giant cell sarcoma character; death occurred in three years; at

autopsy, tumors were found in the internal organs. In a 54 year old woman, the author saw a tumor of round cell sarcoma character, with small, isolated nodules in the lungs. v. Zumbusch saw bone metastases; see also Brutt. The diagnostic difficulties are serious, and the differentiation between sarcoma and "sarcoid" or some other form, perhaps even a tuberculous type of granuloma, cannot always be made on the microscopic preparation alone (see, for example, the case of Vollmer).

For lymphogranulomatosis cutis, see p. 268. For leukemic infiltrates, see p. 204. Concerning the skin affections in leukemic, aleukemic and related conditions, see Paltauf, (lit.), Heinrich, and the critique by Martinotti.

Benign Epithelial Tumors (Epitheliomas).— The nomenclature of epithelial tumors is very confusing. Whereas the author uses the term "epithelioma" merely in the sense of a benign epithelial tumorous growth, others differentiate benign and malignant epitheliomas. For the latter, we recommend that the term carcinoma be used. Yamagiwa chooses "epithelioma" as the name of all epithelial tumors instead of the designation "adenoma" and "carcinoma."

Jadassohn divides benign epitheliomas into two chief groups: (1) those with a known etiology; these are: (*a*) infectious epitheliomas, such as molluscum contagiosum, verruca dura papillomatosa and plana, perhaps also condyloma acuminatum (venereal papilloma); (*b*) chemically produced epitheliomas, thus from paraffin, tar, arsenic, bromine and others; (*c*) mechanically produced, such as callosities, clavus, cornu cutaneum, epithelial cysts; (*d*) actinically produced, such as keratoma senile, xeroderma pigmentosum, Roentgen-ray dermatoses, (2) benign epitheliomas with unknown (predominantly congenital?) causes: (*a*) naevi duri (ichthyosiformes, acneformes, porokeratodes, cornua cutanea juvenilia); (*b*) naevi organomatosi (naevi sebacei, sudoripari, pilosi); under this subdivision are included structures which resemble the normal skin, but because of their situation, are too large or too numerous, or are heterotopic and thus pathological; (*c*) naevi adenomatosi (sebacei, sudoripari); (*d*) naevi epitheliomatosi (syringoma, benign cystic epitheliomas), for these, see Martinotti; "cylindromas," calcified epitheliomas; (*e*) naevi atheromatosi (atheroma, epidermoids, dermoids, milia); (*f*) naevi teratomatosi (teratomas and other mixed tumors).

Adenomas may arise from sebaceous as well as sweat glands, and in their growth resemble more or less these respective types (adenoma sebaceum and adenoma subdoriparum). Both are relatively rare, (lit. in appendix).

The demonstration of a limiting membrana propria around these gland cells differentiates the adenoma from gland-like structures common in certain forms of skin carcinoma.

Both types of adenomas appear usually as subcutaneous tumors, or as warty, nodular prominences. They may reach the size of pigeon's eggs or larger. Adenomas of both the sebaceous glands (Barlow, Jadassohn, Martinotti, lit.), and the sweat glands (Beier, Petersen), may begin as naevi (organ naevi), or even as systemic naevi. Sebaceous gland adenomas (adenomata sebacea) are characterized, even macroscopically, by their delicate glandular structure. The openings of the glands may be seen on the surface. Occasionally, some change into atheromatous cysts. Hyalin degeneration of the vessels and the connective tissue is practically always present, with frequent calcification (so-called calcified epithelioma). The tumors may undergo softening in part, and ulceration. Transitions to carcinoma are met, and may be manifested, even on gross section, by the fact that the adenoma penetrates and ruptures the surrounding connective tissue capsule. Hypertrophies of sebaceous glands are seen in rhinophyma (p. 2105), in the neighborhood of cutaneous carcinomas and in lupus. In many situations, particularly the region of the nasolabial folds, the sebaceous glands are, normally, quite close together.

More marked overgrowth of the lining epithelium of the sebaceous gland acini has also been described, and has led to the classification of a particular type of benign epithelial tumor; dermatologists speak of epithelioma adenoides cysticum (Brooke, Wolters, Kreibich, Martinotti, lit.), or adenoepithelioma of the sebaceous glands (W. Pick). Exact identification of these tumors which appear commonly in the face, is often quite difficult. Krompecher speaks of basaliomas, which contain colloid-cell-cysts similar to the corresponding tumors of the gums and the adamantinomas (p. 590). There also occur, on the face and the head, and the remainder of the skin, quite similar basocellular epitheliomas, parts of which show a cylindromatous-like, cystic structure; these are well known to the author, and have been described by others as epitheliomas (Spiegler), in which, however, the relationship with the covering epithelium or the epithelium of the hair follicles, is still to be demonstrated. E. Hoffmann described such a case in which hundreds of epitheliomas, some of them like tomatoes, appeared on the head, the face and the skin of the body. According to Martinotti, Hazen, there is close relationship to basocellular epitheliomas. As "epithelioma folliculare," O. Israel described lobulated and nodular tumors on parts of the skin covered by hair, which perhaps originated from the hair follicles; in them, he found "coccidia-like" bodies.

The calcified or calcifying epitheliomas have a varied nomenclature as a glance at the literature will show (see, among others, Barlow, Thorn, Walkoff, Leveque and Weissenbach, Landeu, lit.); they are regarded as

endotheliomas, or as calcified sebaceous gland adenomas, as atheromas, or even as carcinomas (the latter by Strassberg and Frey). But they have nothing to do with the latter, as the author emphasizes on the basis

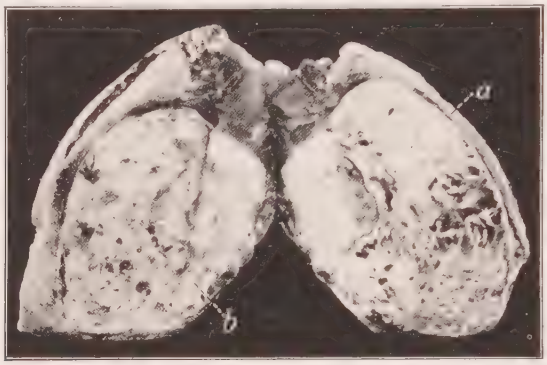


FIG. 1057.—Calcified epithelioma from back, grew in 16 years from the size of a bean to present size; man 60 years old. (Pub. by Murakami, Arch. f. Derm. u. Syph., 1911); a, skin; b, calcified tumor in cellular subcutaneous tissue; shows small hollow spaces; nat. size.

of a number of his own cases (Murakami, lit.). They are rather epitheliomas, clinically benign, the origin of which may be in preformed glands, as well as in deep-lying (dystopic) epithelial cells (Firket), probably from

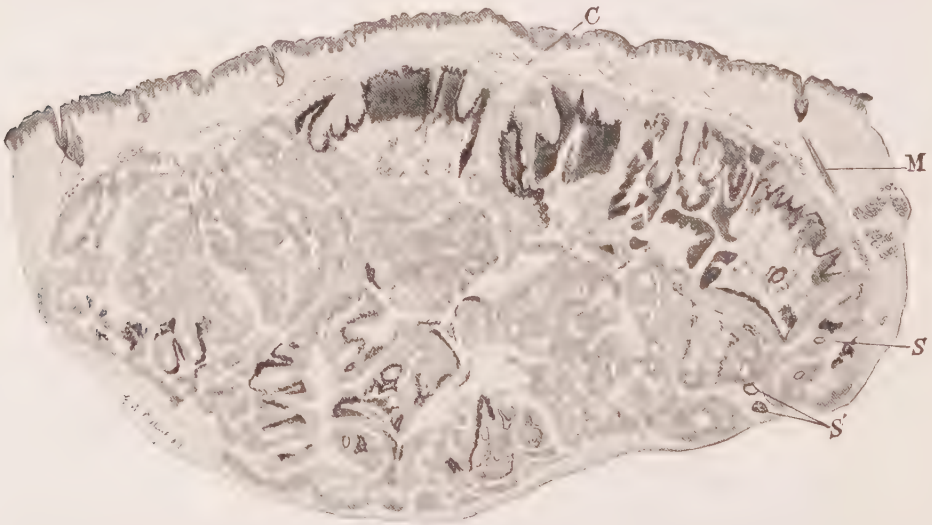


FIG. 1058.—Calcified epithelioma from the subcut. tissue of l. lumbar area from 16 year old mole. The tumor existed for years unnoticed and became as large as a plum. The dark areas in illustr. are calcified. M, an erector pilae; adjacent are numerous sweat glands; c, cystic dilated sweat glands; S, glands like sweat glands. (Magnifying glass.)

misplaced anlage material. They have no similarity at all to the delicate cylindromatous basal cell carcinomas (p. 2202), but occasionally may resemble the epitheliomas with plump prolongations and softening in

the centers of the shreds, as found, *e.g.*, by the author in a tumor from the back of a 61 year old woman; the tumor, reaching the size of two fists, showed no calcification, but on its apex was a small naevus vasculosus. Their situation is in the tissues underneath the lower layers of the skin; they are more or less sharply encapsulated by connective tissue, and are very hard; the smaller types have an earthy, brittle section surface, containing large spaces or numbers of smaller irregular cavities; the larger ones, among which the author has seen one as large as a fist, growing over a period of many years, may contain many pseudo-cystic spaces containing a clear, colorless fluid (cystic calcified epithelioma). They are composed of lobulated complexes of epithelial cells with keratohyalinization and pearls, showing tendencies to necrosis because of poor blood supply, and for this reason, progressive calcification. Between the epithelial masses, are septa of connective tissue which may become ossified (Henzi, lit.). Usually, there is an attempt at the removal of cornified and calcified masses by means of giant cells. The formation of the spaces occurs most probably from liquefaction in the stroma, and the epithelium. Calcified epitheliomas enlarge very slowly. They may occur at any age of life. Among five observations of the author, they occurred three times on the back and once on the thigh (35 year old woman).

The less frequent psammoma carcinomas of the skin should be differentiated from calcified epitheliomas; the former are malignant but have, in common with the latter, the formation of incomplete cornified material, and calcification (Frey).

The sweat gland adenomas, hidradenomas, syringomas (Unna), belong in part to the naevi, *i.e.*, are congenital (p. 2197); they are quite rare, occur most frequently in the face, on one lip, (where ordinarily there are no sweat glands), but have also been reported in many other situations, such as the head, the breast, the skin of the back, the umbilicus (p. 2115), extremities, vulva, etc. In rarer cases they reach quite considerable size, even as large as a child's head; in other cases they produce small papules in large numbers. Usually, they are soft, well outlined, and show small spaces on section corresponding to the dilated gland tubules. Microscopically, they are composed of tubules lined by cylindrical epithelium, in which double layers of the type of coils and excretory ducts can be demonstrated. Consult Pick (lit.), who believes that (*a*) hidradenomas may arise from completed and (*b*) adenoma hidradenoides, from rudimentary sweat glands or from the surface of the epidermis, a division with which Krompecher agrees, and states that the (*b*) type is much more frequent than the (*a*) type. See also Arzt, Kreibich, Rothe, Werther, Coenen, Schwalb, Fischer, Hoffmann and Friboes. See under vulva, p. 1706. Cystic dilatation of the sweat glands leads to a syringocyst

(Neumann), or a hidrocyst (Robinson). These are presented as cystic structures, or like cooked sago granules, as, *e.g.*, on the eye-lids, particularly the lower, (Martinotti). The hidrocystoma tuberosum multiplex (Joseph) is believed to arise from abnormally situated sweat glands (lit. Stockmann). The cystadenoma papilliferum (Klauber, lit.), shows simple and branched papillae inside of the cysts and their excretory ducts, and resembles the papillary cystomas of the breasts, and the ovaries (Krompecher). For sweat gland carcinomas, see Wolfheim, (lit.), Hedinger, (also for the so-called plasmacytomas).

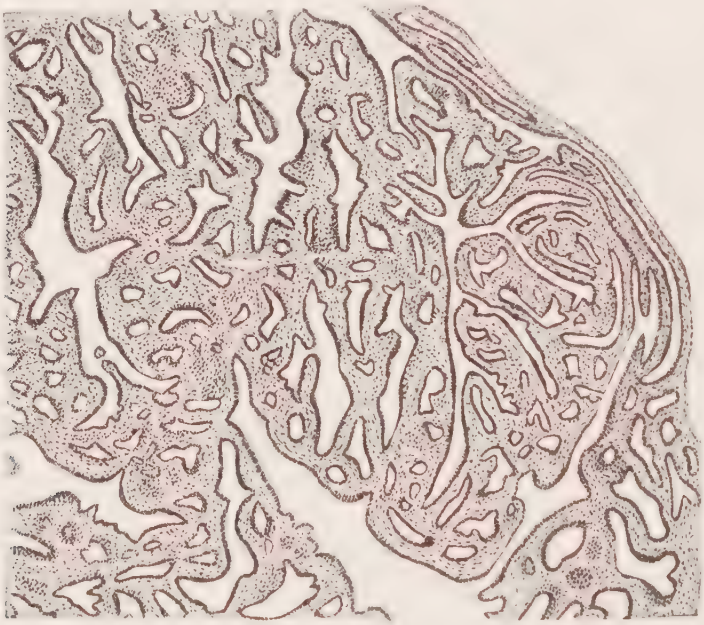


FIG. 1059.—Tubular hidradenoma, from a tumor as large as a pea in the labium majus of a woman 51 years old. (Low power.)

A very remarkable case was observed by the author in the scalp of a 69 year old woman; the tumor, as large as a child's dinner plate, produced a slightly protuberant plateau with an irregular cleft papillary surface; overgrowths from the cystic spaces of a hidrocystoma had broken through the epidermis in a number of situations, so that it was preserved only in isolated places; branched papillae containing stalks rich in plasma cells jutted out on the surface; the stalks were covered by a replacement layer of cylindrical epithelium.

Carcinomas of the Skin

The cornifying squamous cell carcinoma of the skin was formerly called "cancroid," in contrast to the non-cornifying; the former was also

known as a deep invasive, the latter as a flat, superficial skin carcinoma (Thiersch). But this differentiation cannot be carried too far. It is better to speak of a cornifying and a non-cornifying carcinoma. For more details, see p. 2202 and ff. Krompecher described the non-cornifying tumors as basal-cell carcinomas, to which most of the tumors belong described by Braun as fungoid endotheliomas. The author disapproves of the term "cancroid" because, since it is a malignant tumor, it should be

FIG. 1060.—Cornifying squamous cell carcinoma, from the periphery of tumor illustr. fig. 1065. Secondary adhesion of carcinoma to epidermis; a,e pidermis; b, anastomosing squamous epith. nest; c, cornified epithelial pearls; d, scaffolding. (Medium power.)

called carcinoma, and not "malignant cancroid," (see Dreifuss and Bloch), especially not in a histological sense; a cancroid is *eo ipso* always malignant, even though in varying degree.

A skin carcinoma may arise from the covering epithelium, the hair follicles and sebaceous glands, or from misplaced epithelial cells. (For sweat gland carcinomas, see above; melano-carcinomas, see p. 2193.) As a rule, it produces solid cell masses, which, by mitotic division, grow and infiltrate into the neighboring connective tissue. (Ribbert's theory of the origin of a carcinoma states that the reverse occurs, namely, an ingrowth of the connective tissue into the epithelium, the compromised parts of

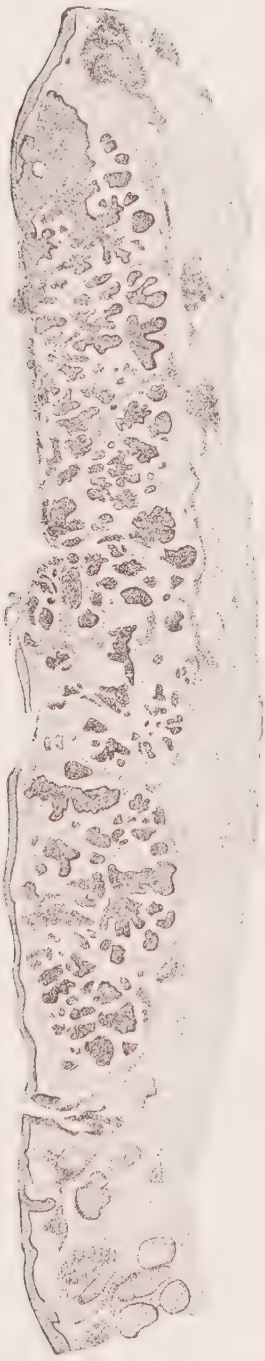


FIG. 1061.—Basal cell carcinoma of nose, but slightly ulcerated; man 63 years old. (Magnifying glass.)

which then penetrate more deeply. It has received no confirmation. Ribbert himself has given up the idea in all but a very few cases. See Geschwulstlehre, page 555, und Das Carcinom des Menschen. The idea lately sponsored by him, viz., that the invasion of the epithelium from above downward through the subepithelial tissues, is produced by subepithelial inflammatory changes, is merely a well known view expressed for many years.) The connective tissue produces alveoli for the carcinoma bodies, or the carcinoma parenchyma. The infiltration varies considerably in the different forms. In the cornifying squamous carcinomas, the malignant skin tumors, it may be particularly well marked, and the islands of carcinoma, injecting the lymph vessels and the tissue spaces, produce an extraordinarily complicated, anastomosing system of prolongations (fig. 1060). On section, there are thus found irregularly rounded foci surrounded by connective tissue, which, however, are connected one with the other (fig. 1060); or such a complete injection of the lymph vessels, that many anastomosing cell plexuses appear.

According to their histology, the various forms of skin cancer may be differentiated into: (1) cornifying squamous epithelial carcinoma and (2) basal cell carcinoma. In the former, the majority of the cells are spinous; they send anastomosing prolongations of various widths into the spaces of the connective tissue, and invade the subcutaneous structures like roots. Lately the terms, spinous or prickle-cell carcinoma, have been used; but the cells may be larger or smaller than the normal cells of the epidermis, and polymorphism may be so marked, that every resemblance to spinous cells is lost; the cells are often markedly fatty. Cornification may be quite atypical. Peripheral cells of the carcinoma masses may even become cylindrical on the borders of the interstitial tissue of the tumor, like the "palisade" basal cells of the stratum germinativum; these are the youngest cells in the foci. Those in the centres of the masses at first

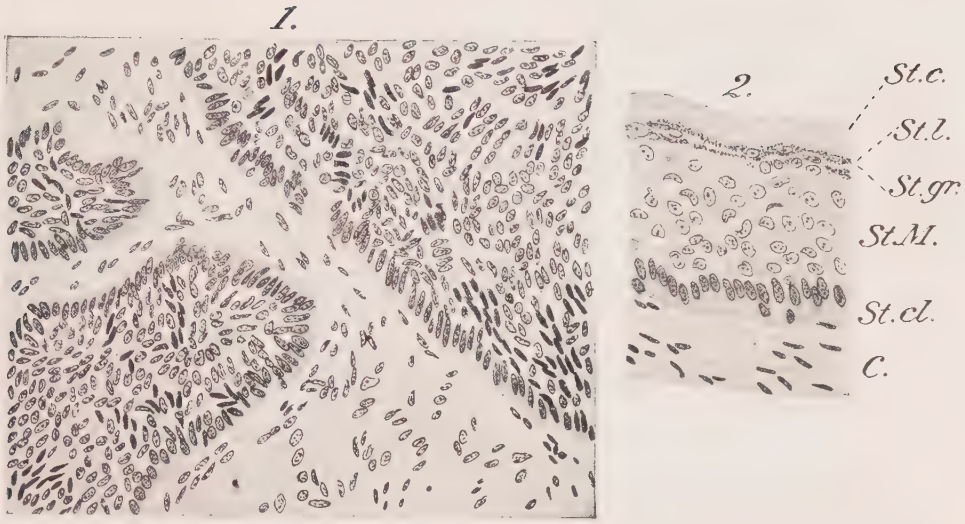


FIG. 1062.—1. Basal cell carcinoma portion. 2, Cross sect. epidermis to show the basal cells of the stratum cylindricum (St. cl) and their similarity to the marginal cells of carcinoma nests. St. M. Stratum Malphighii; St. gr, stratum granulosum; St. C, stratum corneum; St. l, stratum lucidum; c, corium; high power.

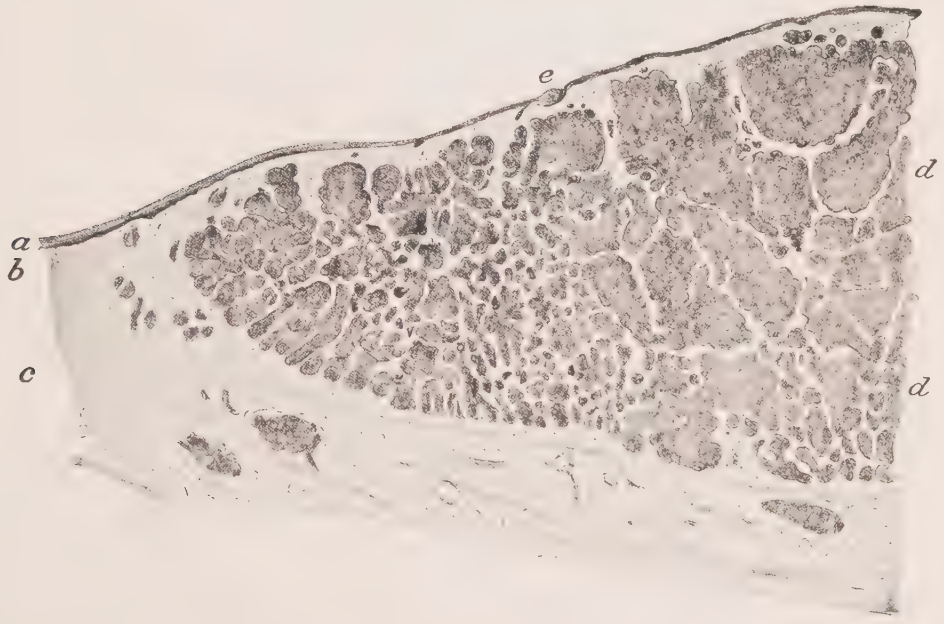


FIG. 1063.—Basal cell carcinoma (Krompecher) gland-like surface epithelial carcinoma from skin of face of woman 60 years old. About ⅓ of the plateau-like tumor is drawn. a, epidermis thinned above the tumor; b, corium; c, subcutis, with connective tissue strands; fatty tissue, blood vessels and isolated sweat glands; d, carcinoma with small lumena in many nests, no cornification; e, epithelial pearl in epidermis. (Magnifying glass.)

contain nuclei, then become anuclear, flattened and fuse in concentrically laminated bodies like onions; the inner layers undergo cornification and form the so-called epithelial pearls; or merely swell, hyalinize and become colloidal masses. The nuclei, still stainable in the concentrically laminated pearls, are parakeratotic. Squamous epithelial carcinomas in which the cornification is wide spread, are called cornifying squamous epitheliomas; the tumor tissues are dry, brittle, white, while the contents of alveolar-like spaces may be pressed out like comedo plugs. Tumors without cornification are called basal cell carcinomas; according to

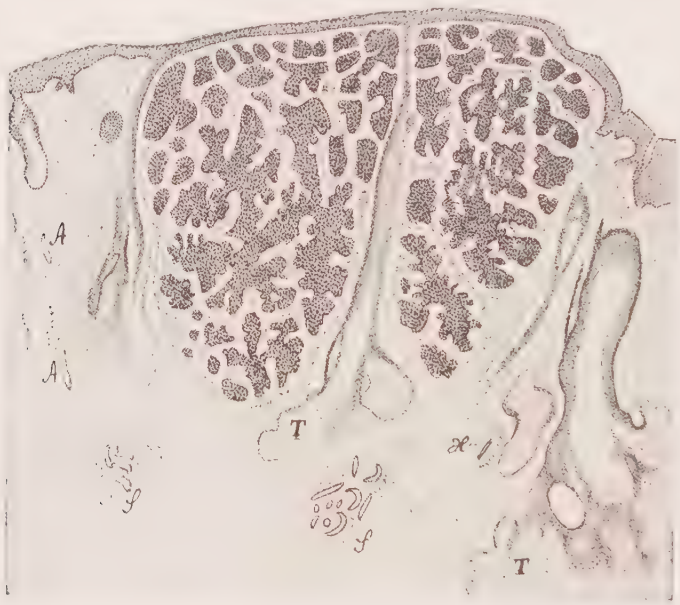


FIG. 1064.—Basal cell carcinoma (adenoid). Recurrence of a warty tumor, removed several years before from face; several hair follicles T; and two sweat glands; H, cross section of hair. A, duct of sweat gland; man 45 years old. (Med. power.)

Krompecher, these do not become further differentiated, but Ribbert, who calls them "adenogenous" carcinomas, states that they become dedifferentiated, *i.e.*, descend to a simpler developmental stage (pp. 622, 641, 1673). (Cylindrical epithelial mucous membranes, see pp. 697, 819, and Krompecher.) This particular group is believed to arise from the cylindrical cells (basal cells) of the stratum germinativum, also from the basal cells of the accessory glands of the skin, *i.e.*, their excretory ducts; because of their gland-like structure, they are frequently called glandular surface epithelial carcinomas (Krompecher). These are the tumors which macroscopically are fungoid, plateau-like, or ulcerated; they were previously called plexiform sarcomas, or endotheliomas (Braun, Borst and others). Borrmann considers them as originating from mis-

placed epithelial masses in the corium, or, unfortunate hair and gland anlage, and calls them (histiogenetically) corium carcinomas. This designation, however, seems too indefinite histologically, and should therefore be abandoned. It cannot be doubted that some of the basal carcinomas do come from such misplaced elements, but it must be recognized that the same types of carcinoma may arise from mature basal cells. When, however, Ricker-Schwalb call the basal cell tumors, in general, sebaceous and sweat gland growths, they deserve the criticism of Krompecher, since these basal cell carcinomas have merely a very distant

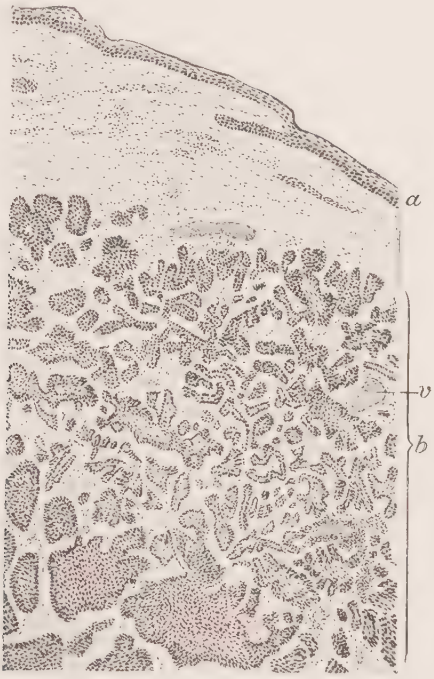


FIG. 1065.—Basal cell carcinoma (Krompecher) from cheek of a man 70 years old; portion near margin of a coin-shaped tumor, ulcerated in the middle. a, epidermis; b, corium with carcinoma; v, vein. The connective tissue shows hardly any reaction. (× about 40.)

resemblance to the notorious adenomata sebacea and syringomas; they are not characteristic sebaceous nor sweat gland tumors, but rather incomplete attempts to produce skin glands (Adamson). On the other hand, basal cell carcinomas may originate from the epidermis, become free of this structure, and without cornification, spread in the corium. But then basal cell carcinomas, which arise in the corium, may secondarily be related to, or communicate with, the epidermis. As Kyrle emphasizes, the epidermis, *i.e.*, its basal layer, may be the matrix for the formation of post-fetal sebaceous glands, as well as the point of origin of the covering epithelium and of the cornified material. Krompecher lays great stress

on the term, basal cell carcinoma, since these tumors always retain their basal cell character, and do not undergo further differentiation into spinous cells; later, he uttered the warning, that this terminology means nothing more than the morphology, because histiogenetically considered, all skin carcinomas arise from the stratum germinativum, *i.e.*, the basal cells. With this reservation, we may therefore call most of the non-cornifying skin carcinomas, basal cell carcinomas, remembering that there

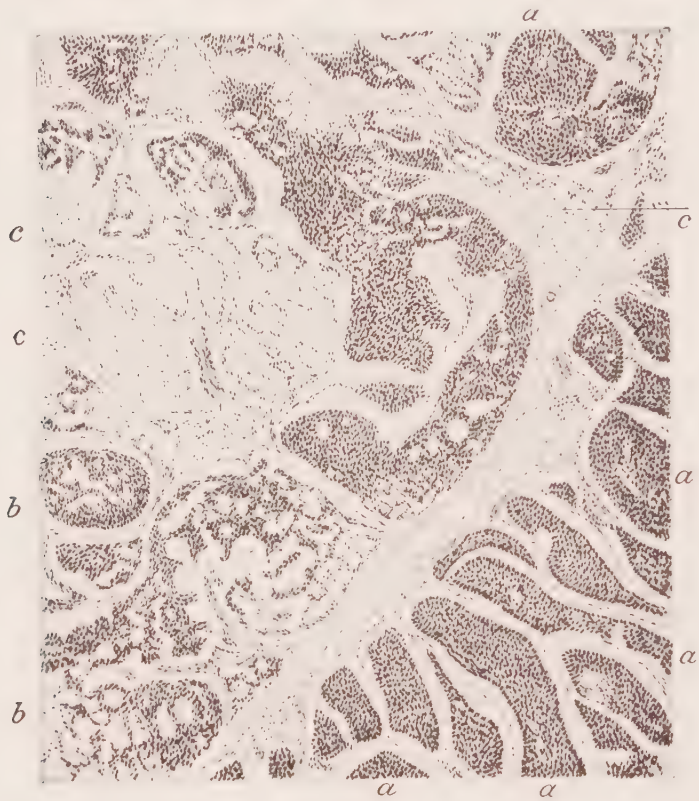


FIG. 1066.—Basal cell carcinoma with marked hyalin and mucous metamorphosis of stroma and less so of the carcinoma nests (so-called cylindroma-formation). Tumor of right temporal area of woman 62 years old; existed about 3 years, growing faster toward the last. At a, typical larger nests surrounded by hyalin bands, showing several small hollow spaces; b, marked hyalin-mucoid metamorphosis; c, barely a few connective tissue strands remain, all else liquefied (× 78).

do occur squamous cell epitheliomas without cornification. The designation is morphologically too narrow perhaps, inasmuch as, in addition to the obvious "Krompecher-carcinomas," there occur others in which the epithelial cells are no longer cylindrical, but have an indifferent spindle-shaped contour, so that they resemble the cells of a spindle cell sarcoma; the cylindrical character is, however, retained here and there, particularly among the cells in the edges of the plugs (figs. 1063, 1065 and 1066).

But since this is often true of the border cells, *i.e.*, the youngest cells of a cornifying squamous carcinoma, it is morphologically improper to speak of these as "prickle-cell carcinomas;" cornifying squamous carcinoma is a better name.

The histological pictures of basal cell carcinomas ("Krompecher-carcinomas") are quite varied. In some, there are solid strands and nests, which are branched, and produce bulbous or pointed prolongations. The strands are plump or delicate, and usually show cylindrical cells along their edges, while the majority are spindle-shaped, sarcoma-like forms, or the cylindrical cells are entirely absent (figs. 1063, 1065, 1066). In other tumors, spaces and cystic necrotic masses occupy cavities in

FIG. 1067.—Basal cell carcinoma in area of malar bone of older woman; tumor much ulcerated; a, connective tissue stroma with blood vessels; b, strands consisting of spindle-shaped cells; c, with hollow spaces; d, with necrosis; e, concentric arrangement of squamous cells (low power).

the foci, so that many plugs contain innumerable, small rounded or oval spaces, while larger plugs are reduced to merely a wall of well-staining cells. Macroscopically visible cysts may appear, sometimes in large numbers, (cystic basal cell carcinomas). Two things must be kept in mind: the spaces have arisen from destruction of the carcinoma plugs, or there has been hyalin or mucoid change in the stroma, which is frequent in many of the so-called cylindromas (fig. 1066). Or the epithelium may grow in isolated bands of cylindrical cells, in garlands, or gyrus-like folds. Less frequently, quite delicate pictures consist of thickly grouped, more or less convoluted and contorted, epithelial tubes, some containing lumena and resembling sweat glands scattered among solid foci and nests. Naturally, it is not proper to speak off-hand of these tumors as sweat gland carcinomas. Krompecher himself differentiates solid, cystic, and adenoid types (on squamous epithelial surfaces and cylindrical cell

mucous surfaces). As he also emphasizes, there appear, even though rarely, combinations of cornifying and basal cell carcinomas; the author has repeatedly seen these tumors (Clairmont); the cells of a basal cell carcinoma have undergone further differentiation. Korbl saw basal cell carcinoma recur as a cornifying tumor; indeed transformation to a cornifying tumor is shown very frequently in the basal cell carcinomas treated by x-rays. This short synopsis by no means exhausts the forms these tumors may take. We will still mention a large "plug" form, in which extensive necrosis and liquefaction occur in the prolongations. This was formerly believed to be an endothelioma; such types occur particularly often in the os uteri (fig. 1067).

The connective tissue, *i.e.*, the supporting framework of the carcinoma, is without particular change, or may be hyalin and tough, or overgrown; infiltrations are frequent by leucocytes, lymphocytes, or plasma cells (lit. by Prytek); the latter sometimes appear in almost tumor-like masses, but mean, in general, slow growth, and, according to Unna, relative benignity (?). Occasionally, many eosinophilic cells are found in the inflammatory reaction zone around the tumor (Feldbausch, W. Fischer and others; extensive lit. by E. Schwarz; Weill). The elastic fibers are pulled apart and surrounded by the actively invading epithelium, but are sometimes still demonstrable in the midst of tumor nests; according to Neuber, there is also an active increase in their number. They disappear where there is severe inflammatory reaction in the interstitial tissue (Zieler), but a mild round cell infiltration does not damage them. Collagenous fibers are continuously renewed in carcinomas. In many cases the interstitial tissue produces the walls of "alveoli" for the carcinoma bodies, from a luxuriant growth of granulations, which later become richly cellular connective tissue, or tough scars. The more marked the overgrowth, the more marked the prominence of the tumor, and when the interstitial tissue is very richly cellular, it may be of soft consistency; this also helps produce the papillary forms of squamous carcinoma in those not arising from a previous papilloma. Inflammatory processes are very frequent in the tissue of the carcinoma itself, and may produce much suppuration; they usually originate from external accidents. The suppuration extends into the interstitial tissue, and even liquefaction occurs of large parts of the tumor. A carcinomatous ulcer is developed which, in the case of cornifying carcinoma, has tough, infiltrated walls and a tough infiltrated base. Fungoid granulations containing carcinoma cells may grow from the base. But the ulceration of basal cell carcinomas is usually superficial, and related to the superficial, and slow spread of these tumors.

The granulation tissue around cornifying carcinomas often contains numerous large polymorphous cells, surrounding the pearly bodies of

foreign body giant cells, and may even invade them, so that the pearls become brittle; they may become filled, partly at least, by granulation tissue. Becher, Petersen, Schwarz, speak of this as spontaneous healing; Borrmann, Delamare and Lecene, emphasize that the phagocytic ability of these giant cells is exerted only on material already dead. By considering not only the stromatogenous giant cells, but also the granulation tissue which grows into the carcinoma masses, and occasionally isolates entire foci, it may be thought that this is a healing process, analogous to that spoken of in the carcinoma granulomatosum of the stomach (p. 699, Ribbert). But if, on the other hand, merely giant cells are present, it is quite possible, as Orth suggests, that regressive changes in the carcinoma cells are the stimulus for the further progress of the carcinoma; foreign body giant cells, and partial destruction by granulation tissue, are of little significance. Not rarely, particularly in squamous carcinomas, the giant cells are of epithelial origin; they have gigantic chromatin-rich nuclei, or a number of nuclei clumped in a mass. The finding of giant cells has led to the erroneous assumption of a combination with tuberculosis (Petersen). Fatty degeneration is very common, as is hyalin change in basal cell carcinomas; the latter produces spheres and strands in the epithelial shreds, but more frequently the hyalin is in the connective tissue; both conditions produce a carcinoma cylindromatosum (fig. 1066). Microscopic calcification is also not uncommon in isolated cornified epithelial nests; many foreign body giant cells collect around the petrifying masses. Macroscopic calcification of true skin carcinomas is very rare (compare calcified epitheliomas, p. 2197).

Etiology.—Many skin carcinomas follow chronic (chemical) irritation (*e.g.*, in chimney sweeps, workers in tar and paraffin, p. 1540; the experiments of Yamagiwa and others), arsenic keratoses, (p. 2166), or actinic (Roentgen) dermatitis; the term "precarcinomatous" is often applied to certain chronic eczemas, the melanodermia of old age, xeroderma pigmentosum (Nobl, Harzen), etc.; multiple primary carcinomas have appeared under these circumstances. Carcinoma may also follow treatment with radium; an extensive cornifying carcinoma followed radium treatment of a naevus flammeus in a 9 year old child. Others appear in scars, particularly in those of lupus, but also in others, *e.g.*, the scars of decubitus of the sacrum (Trendelenburg), or operative scars, or in an old gun-shot wound (Melchior); they develop particularly readily in slowly healing burns, or their scars. It is quite remarkable how late the carcinoma (usually a cornifying tumor) may develop in a scar; thus in a 53 year old man, the author saw a cornifying carcinoma of the back of the hand in the region of a scar from a burn received in childhood; in a 64 year old man, there appeared a cornifying carcinoma as large as a dollar, in the thigh, in the

scar of an osteomyelitis dating back to childhood. In other cases, carcinomas appear in chronically irritated, purely traumatic ulcers, or in bed-sores. The author examined the amputated lower extremity of a 28 year old girl (Dr. Methner), who had suffered extensive atrophy of the leg following an injury by a machine in her fifteenth year; the extremity could be used with the help of a support for the knee. A year of pressure from this apparatus, produced a bed-sore, just above the internal condyle of the femur; this sore soon presented itself as a hard edged, ulcerating cornifying carcinoma, one centimeter thick and as big as a saucer; it contained innumerable giant cells around and among the carcinoma cells. There was marked infiltration of the corresponding inguinal lymph nodes. Furthermore, carcinomas appear in leg ulcers, (even bilaterally), occasionally, as in the observation p. 167, after it has been present for decades; it has appeared in a perforating ulcer of the foot (Zahn and others), in elephantiasis, in old healed bone fistulae lined by epidermis, etc. The author saw a squamous cell carcinoma (Dr. Methner, Breslau) in a 53 year old man, originating in a fistula present for 34 years on the outer side of the left thigh; the fistula had been produced by an osteomyelitic central necrosis, the sequestrum of which was not removed; the carcinoma had arisen only during the last year; the inguinal nodes were markedly involved. (See also the case of Rehn and von Retzlaff, a cornifying carcinoma arising in the scar of a forty-seven year old fistula following a gun-shot wound in the thigh.) Carcinomas also arise in specific inflammatory processes; to these belong the lupus carcinomas, and the carcinomas of gummatous tissues. They may originate from warts, particularly the senile types, in condylomas, or combined with cutaneous horns; the latter are rare. Atheromas may become carcinomatous (fig. 1071); ulceration almost invariably precedes the process. Subcutaneous skin cancers not infrequently appear in germinal anlage fistulae (p. 330, branchiogenic carcinoma). Many skin carcinomas are obscure in their origin. They are occasionally multiple in the same region, *e.g.*, the face, the ears, the scrotum. In a case described by C. Kauffmann, it was assumed that the carcinoma was inoculated from the back of the hand to the conjunctiva. At the autopsy of a 71 year old woman, a cutaneous carcinoma of the face, and one of the lower abdominal region, were found by the author. Skin carcinomas prefer older individuals, but the author saw a rapidly recurring tumor behind the ear of a 16 year old girl; from a 16 year old boy, he examined the fourth, and this time, inoperable, recurrence which had invaded from the left eye into the region of the neck as a squamous carcinoma; it had arisen two years previous from a small wart which the patient himself had pulled off. Roentgen ray carcinomas (p. 2084) usually occur in younger individuals.

According to the classical investigations of Thiersch, two clinical forms of cutaneous carcinoma may be differentiated: a shallow and a deeply invading form, which, however, cannot always be sharply separated, because there are transitions. A third form might be called the papillary variety.

The flat cutaneous carcinoma, histologically usually of basal cell type, occurs in the face, the cheeks, forehead, the nose, the nasolabial folds, the eye-lids, etc., most often in old age. The beginning may be expressed as a seborrhoea (seborrheic keratosis). The basal cell carcinoma often arises multicentrically. The early tumor appears as nodules in the skin or the subcutis, which is then destroyed, leaving an irregularly rounded, shallow, dry ulcer, surrounded by a quite small hard rim, or a nodular wall. The tumor resembles an ulcer so closely that even today the term "ulcus rodens" is used. Occasionally the tumor grows upwards, the base of the ulcer reaches the level of the skin, or is even raised as a plateau with sharply falling, or somewhat overhanging, hard edges (ulcus elevatum). Frequently, it undergoes cicatrization in certain parts, is covered with epidermis, and appears healed, but the ulceration slowly progresses at the periphery. Not rarely, it shrinks until the skin in the neighborhood is pulled in with radiating folds. The flat, cutaneous basal carcinoma is relatively benign in comparison to the cutaneous carcinoma which invades the depths. It must be remembered, however, that there are frequent fungoid tumors with little tendency to destruction, likewise of the type of basal cell carcinoma.

Occasionally, primary multiple basal cell carcinomas are observed; in a 54 year old man, the author saw three distinct basal cell carcinomas in the face (temple, right and left cheeks).

The course may be very chronic; it may last many years, until the tumor reaches considerable size and depth. Its restricted malignancy is expressed in many cases by merely local recurrences after operative removal. Recurrences may never appear. Metastases usually do not occur. Concerning the general benign prognosis of basal cell carcinomas in comparison to squamous cell epitheliomas, see Marassowich. The so-called flat cutaneous carcinoma occasionally invades the depths (in the face to the skull and dura), and by a sudden rapid growth destroys a large amount of tissue, particularly of the face; in such cases it is possible that there has been a transition into a cornifying squamous tumor (p. 2208).

The regional lymph nodes are seldom infiltrated, but they become hard and slightly enlarged. A particular type of superficial carcinoma is Paget's disease of the nipple (p. 1787).

The depth-invading cutaneous carcinoma is histologically of a cornifying squamous type. To this class belong many tumors of the lips, the

large majority of the extremity carcinomas, and many ear carcinomas; these, in contrast to the basal cell carcinomas, are endowed with malignant properties. They begin likewise as nodules with flat ulcerations, but from the very first the malignant character is quite obvious, inasmuch as the masses spread in all directions into the depths, as well as toward the peripheries. Thus there soon appears a nodular, fairly moist, medullary tumor, the surface of which is more or less deeply ulcerated (fig. 1068).

In some cases the ulcer is shallow, very irregular and angular, in others it is deep and crateriform.

By fatty, ulcerative, or caseous destruction of the tumor tissue, the skin covering the underlying tumor is perforated by many holes, so that

FIG. 1068.—Immense cornifying squamous cell carcinoma with surface ulceration of back of r. hand of woman 71 years old. Tumor existed for 1¼ years; pain for 9 mos.; edema of fingers; axillary nodes had become mushy; amputated.

FIG. 1069.—Fungoid carcinoma on r. fourth finger of man; cornified; nat. size.

there is considerable resemblance to a perforating gumma. The author saw such a condition in a 36 year old woman, as a colossal carcinomatous ulcer of the popliteal region. The invading carcinoma always grows much more rapidly than the flat type, it infiltrates the lymph nodes more quickly so that they enlarge to mighty tumors with a tendency to softening; it also produces much more frequent metastases in the internal organs, particularly the liver.

Infiltration of the underlying bones and spontaneous fractures are common in the deeply invading lip tumors, (lower jaw), or in those originating in a leg ulcer; the author saw it occur in a lupus scar carcinoma of the upper arm. Enormous, deeply invading, cornifying carcinomas of

flat spread, and a number of centimeters height, with fungoid prominences, occasionally arise in the back of the hand of older persons (fig. 1068); the author saw these types in a 74 year old man, and an 85 year old woman; quite similar tumors occur on the dorsum of the foot.

The **papillary cutaneous carcinoma** (fig. 244, p. 567), is characterized by papillae on the surface, while the epithelial overgrowth penetrates below. It originates in a papillary fibroepithelioma (see the observation of the author in the chapter on the vulva), or as an independent growth. In many situations (lower lip, nose), the surfaces of the papillae are so markedly cornified, that compact masses resemble cutaneous horns, and thus induce mistakes in the diagnosis (H. Burckhardt). In other situations, *e.g.*, the penis (see there for papilloma), branched papillary overgrowths look like cauliflowers. The benign prestage may be an acanthoma, or verruca papillaris; the spinous cells predominate en masse, and in recent dermatological literature, the expression, warty spinous cell carcinoma (Hazen uses the expression in a histiogenetic sense), or spinocellular carcinoma is used. The author has not realized the necessity for such a nomenclature. The designation, cornifying squamous epithelial carcinoma, or, cornifying carcinoma, seems sufficient.

See pp. 2188 and 2193 concerning naevo-carcinomas and melanocarcinomas; among them, there also occur cornifying squamous epithelial carcinomas (Bekey).

Secondary cutaneous carcinomas vary histologically according to their points of origin. They are particularly frequent from breast tumors, carcinoma of the uterus (lit. Offergeld), or the stomach, the large intestines, (when they adhere to the anterior abdominal wall, usually regional, seldom widely distant, as for example, on the lower thigh; Neustadt). Stropeni saw them in lung carcinomas, the author, in the lower abdominal region from thyroid carcinoma. (Daus, Montier and Marre, Kauffmann-Wolf, Fillie, lit., Kreibich.) They are often localized around the umbilicus, as, *e.g.*, in primary gastric carcinomas (Quenu and Longuet). Concerning metastatic lymphangitis carcinomatosa of the skin with local edema, see Geipel; concerning lymph vessel carcinosis and general edema, see Schierge. The relation of the skin nerves inside of metastatic tumor nodules is mentioned on p. 2031. For carcinoma in the canals formed by puncture wounds, also in cutaneous scars of the abdomen, see p. 882.

Atrophy of the Skin

In senile atrophy, the skin is thin, the papillae of the cutis are reduced in size, and focally disappear.

The atrophic fatty tissue takes on a dark yellow to brown color; instead of the darker fat droplets, there are final brown pigment granules.

Concerning the easily solidifying, colorless fat of small children, and the oily yellow fat of adults (lipochrome), see p. 2170 under fat sclerema.

According to M. B. Schmidt, the elastic fiber network in the poorly cellular cutis is retained longer than the connective tissue bundles (collagenous fiber bundles; collagenous atrophy). But the elastic fibers are more convoluted, pushed more closely together, swollen, coalesced and then hyalinized and disintegrated to fine granules. The smooth muscle fibers undergo granular swelling and hyalin degeneration; their nuclei become deformed and show chromatolysis (Vignolo-Lutati). Some vessels are destroyed, some are dilated; they are frequently hyalin or fatty, or calcified. The rete Malpighi is thin; the horny layer is thus closer to the papillary bodies, and is dry, brittle, and scaly (pityriasis simplex). Pigment deposits in the form of yellow or brown granules are found in the corium and in the rete. The hair follicles shrink, the sebaceous glands undergo atrophy and disappear; the sweat glands are similarly affected. The hair falls out, and as a replacement, there is found perhaps a lanugo hair (alopecia senilis). When the openings of hair follicles are filled with desquamated epithelial masses, the retention cysts resulting contain numerous lanugo hairs.

Particular forms of degeneration of the skin are: the so-called colloid degeneration, or colloid milium, caused by local degenerations of the collagenous tissue under the influence of light and air, the term "weather-beaten" describing it. Pseudoxanthoma elasticum of obscure genesis, is a local destruction of elastic fibers, perhaps following previous increases (lit. Dohi). The site is frequently the flexor surfaces of the larger joints; it is combined, as a rule, with yellow discoloration. In so-called idiopathic skin atrophy, or better, dermatitis idiopathica atrophicans, since chronic inflammatory changes (endo- or exogenous causes?), precede the atrophy, the skin becomes similar to rumpled cigarette paper (Rusch); other signs are fibroma-like foci and stripes, baldness of the parts, more rarely, mutilations from annular constrictions of the toes and fingers, and still more rarely, torpid ulcers. Dermatitis maculosa atrophicans (Heuss, lit.), is a variation of dermatitis idiopathica atrophicans, and is occasionally characterized by tumor-like saccular formations of the swollen connective tissue; it may perhaps be confused with neurofibromatosis. Concerning maculae atrophicae in syphilis, see p. 2134.

Marantic atrophy occurs in cachectic individuals, and is characterized by thinness and dryness of the skin, often accompanied by desquamation of tiny scales (pityriasis tabescentium).

Stretching atrophy occurs on the abdomen and the thighs and leaves striae (striae distensae cutis). These stretching stripes are blue red, then white, of varying, cicatricial appearance; the papillae are flattened, and the

stretched and shredded connective tissue bundles of the cutis, previously arranged in a network, become parallel and thinned.

Whereas Langer assumed there was merely a distensio cutis in the actual sense of the word, Schlee also described minute lacerations of the cutis. According to Troisier and Menetrier, the content of elastic fibers and vessels inside of the striae may be significantly reduced. Heuss believes the disappearance is merely a seeming one, but tears and destruction have been demonstrated (Unna); according to Zieler, new formation may follow tearing.

The change occurs most frequently during pregnancy (striae gravidarum), also in edema, in marked adiposity of the panniculus, etc.

The stretching stripes in typhus are known as striae patellares because of their particular length. They lie in the skin above and over the patella, at right angles to the long axis of the leg, also along the outer surfaces of the thighs. They are rare, and may occur in other severe diseases, such as dysentery, endocarditis, scarlet fever (Bleibtreu), osteomyelitis, etc. The cause is an overstretching of the skin which has become less resistant; a position with bent knees favors their origin. It is also thought that there may be a growth asymmetry of the soft parts in relation to the growth of the bones of younger individuals, in analogy to the so-called cutis verticis gyrata, a peculiar skin folding on the scalp—lit. by Adrian and Forster—a consequence of incongruent disharmonic development of the scalp in relation to the underlying bones. They are first dark red, and later white. As a sign of previous serious sickness, they may probably have practical interest in later life, perhaps even after many years (Kaiser); lit. Tauber, Bunch.

Cutis laxa s. hyperelastica is an increased elasticity of the skin (in the so-called skin human; hereditary, see Furnrohr); folded skin (chalodermia), is the development of folds on the relaxed skin of the abdomen and the gluteal regions (Ketly). According to Froriep, the skin loses its elasticity during cholera, etc., and folds, lifted upward, may remain in their position for five minutes or more, before they are again smoothed out.

Concerning the diseases of the nervous system in relation to skin atrophy, and concerning skin atrophy in general, see lit. by Finger and Oppenheim.

Amyloid change in the skin (Schilder, lit.), is often found in general amyloidosis; it prefers the axillae, and the scalp. The sweat and sebaceous glands disappear in the face of the degeneration. The amyloid is situated between the epithelial cells of the sebaceous glands, and the elastic fibers which surround them; it also invades the elastic fibers of the muscle layer of the sweat glands, even into the connective tissue covering. In the hair follicles, the amyloid lies between the epithelium and the elastic fibers

which normally are closely applied to the cells. In the papillary bodies, it may be in the form of "amyloid bodies." The cutaneous connective tissue is also infiltrated (Konigstein).

Changes in the Sebaceous Glands, Sweat Glands, Hair, Nails

Sebaceous Glands.—The sebaceous glands (glandulae sebaceae), with few exceptions (in the labia minoris, preputium or the glans, the skin of the areola, and the red edges of the lip, see Schaffer), as true hair follicle glands, appear in the larger hairs as appendages of the hair follicles; in the woolly hairs, on the other hand, this is reversed, inasmuch as the woolly hair follicle is an appendage of the enormously developed sebaceous gland (Stoehr, Lehrb).

Too much secretion on the part of the sebaceous glands is called seborrhoea. It consists of sebum composed of fat and destroyed cells. If the proportion of fat is large, it is a seborrhoea oleosa; if dry, scaly conglomerations are formed on the skin composed of dry sebum and cornified epithelial cells, the condition is known as seborrhea squamosa. From external dirt, the scales become gray or black.

Concerning the skin sebaceous secretions, see lit. by Kuznitzky.

Seborrhea may be local or general; the latter is found in the newborn, and may lead to ichthyosis sebacea (p. 2171). Local seborrhea occurs particularly on the scalp (seborrhea capillitii), in the face, and the genitalia. In children, the so-called gneis is the product of a seborrhea capillitii; it is composed of fat, dirt, desquamated epidermis and hair, and develops, according to Kaposi, as a remnant and progression of seborrhea and extensive regeneration of epidermis, as met in the fetus and the newborn over the entire body (vernix caseosa). Czerny believes that gneis is a symptom of an exudative diathesis (p. 260). In adults, seborrhea capillitii is most frequently a continuous desquamation of small scales (pityriasis capillitii) as a result of inflammations, *e.g.*, erysipelas, or more frequently, as a spontaneous condition. The hair may fall out (defluvium capillorum), and baldness appear (alopecia pityrodes s. seborrhoica s. furfuracea). Other sites of election are the umbilicus, the genitalia.

Diminution of sebaceous secretion (asteatosis cutis s. xeroderma), is rarely idiopathic, but usually concomitant with other skin diseases such as psoriasis, ichthyosis, elephantiasis, prurigo, lichen ruber, leprosy, etc. The skin becomes dry, chapped, and occasionally shiny (pityriasis simplex).

Mechanical obstruction to emptying. When egress of sebaceous material is prevented, *e.g.*, from drying in the excretory ducts, the thick secretion in the ducts and in the glands themselves produces small cystic retention tumors.

Comedos.—A black spot represents the opening of an excretory duct. By exerting pressure on either side, a vermicular structure is pressed out, consisting chiefly of sebaceous material with some cornified epithelial cells and bacteria, and more or less of woolly hairs. The acarus s. demodex folliculorum is also found frequently in the secretion, chiefly, and almost in every human, in the hair follicles of the nose, (according to Du Bois, who found it from the fifth year of life onward). Von Raehlmann was mistaken when he believed that the acarus was the irritant of diseases of the eye-lid; see Hunsche, and lit. by Gmeiner. (See also acarus in the mamilla, p. 1803.) The comedo is therefore produced by a collection of secretion in the excretory ducts of the sebaceous glands, or in the communal excretory ducts of these structures and a hair follicle; it is a sort of miniature atheroma. Sites of preference are the face, particularly the angles of the nose, and the pinna of the ear.

When inflammation is added in the walls and the neighborhood of the hair follicles and sebaceous glands (staphylococci)—a frequent enough occurrence,—a small red papule with a black center point is found (acne punctata); an acne pustulosa is produced if the nodule undergoes suppuration. This acne, as is well known, is very frequent during the years of puberty (Riehl).

Milium is a small, white, spherical shaped skin nodule covered by corium layers, papillae and rete, produced by a collection of epidermis cells in one or more lobules of a superficially situated sebaceous gland. Where the sebaceous gland empties into a hair follicle, the milium produces dilatation of the hair follicle itself (Kaposi). Sites of preference are in the skin of the eye-lids, the cheeks, forehead, on the inner surface of the labia minora, etc. On opening with a pin, a white body, smooth or nodular, composed of epidermoid cells, may be pressed out.

Concerning pseudomilia, and other epidermis cysts following other dermatoses, see Martinotti (lit.).

Sebocystomatosis is described by Gunther as a condition in which transparent cysts, containing an oily substance, of the size of pepper grains, shine through the skin; they are lined by one to two layers of laminated epithelial membrane, and are related somehow to the sebaceous glands. According to Klausner, these multiple follicular skin cysts depend upon a congenital hereditary disposition of the follicle to the formation of cysts.

In speaking of atheroma, it is well to differentiate several different structures for which the conventional term atheroma is used:

(1) Simple retention cysts, larger, becoming as large as chestnuts, usually arise from hair follicles, or, if smaller, from sebaceous glands; these may be called hair follicle cysts, or sebaceous gland cysts, or briefly,

follicle cysts (Chiari); these are the true or simple atheromas. The snaring-off usually occurs on the surface, thus, from eczema and scar formation; in the course of time, they may be subcutaneous, and completely independent of the epidermis. On the scalp, these retention cysts are made more obvious when the hair falls out. They are often multiple, and reach the size of chestnuts and larger.

The lining of the cysts consists of two to four layers of flattened epithelial cells. The neighboring connective tissue capsule is poorly vascular and without papillae.

(2) Actual new growth atheromas, from congenital or traumatic misplacement of the skin, or, more frequently, only of the epidermis, produce dermoids and epidermoids, the walls of which are composed of the entire skin, or merely the epidermis. They are solid tumors, chiefly

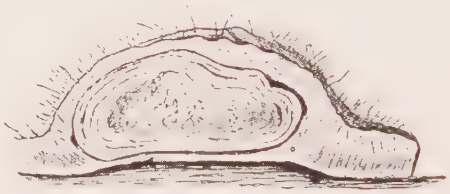


FIG. 1070.—Dermoidal atheroma.

of laminated desquamated epithelial cells; on section they are dry, or, if the epithelial masses undergo liquefaction, moist, and there is a cystic cavity containing a pulpy substance.

The lining may be built up of a number of layers of epithelium, as in the epidermis. The connective tissue capsule is richly vascular, and may show papillae.

If the pulpy consistency of the content is taken into consideration, the expression dermoidal and epidermoidal atheroma may be used in contrast to simple atheroma, *i.e.*, the follicle cyst. Since these dermoids do not progress in contrast to others, *e.g.*, in the ovaries and testes, Chiari calls them dermoid cyst atheromas. The more correct designation would be dermoidal and epidermoidal atheroma cysts. But the nomenclature of this subject is almost hopelessly confused. The same is true of the question, of whether the frequent hair follicle tumors of the scalp are retention cysts, or are, in general, epidermoids, as believed by most dermatologists (lit. by Hesse, Jadassohn, Frei).

Follicle cysts as well as epidermoid and dermoidal atheromas with pulpy content are macroscopically quite similar, inasmuch as the hair follicle encloses a pulpy mass, which occasionally shows a laminated leafy structure.

The content of follicle cysts and dermoidal atheromas consists of cornified epithelial cells, fat droplets, detritus, often cholesterin, and not rarely, fine hairs. It may be quite viscid, like honey, if it contains much fat. In the beginning, the content of epidermoid cysts is coagulated, and consists of cornified epithelial cells; it later becomes pulpy when detritus, cornified cells, cholesterin crystals are added, but it never contains fat. The inner surface of the wall of epidermoids is composed of a varying number of thick layers of flat epithelial cells. Occasionally papilla formation occurs on the walls (Zimmermann). In retention cysts the inner surface is, as a rule, smooth, and covered by a number of layers of thin epithelial cells.

Traumatic epidermoid cysts are called by Reverdin, cystes epidermiques, by Sutton, implantation cysts, by Garre, epithelial cysts; they may reach the size of hazel nuts, and occur particularly on the volar side of the hand, and on the thumbs. They often follow an insignificant injury which may be overlooked (Klein), but in which there was implantation of skin or merely of parts of the epidermis. In the first case, cysts occur, in the other case, only isolated large epithelial pearls. In a similar way, after trauma of the hand, there may occur misplacements, or projections of very small pieces of skin (epidermis with the underlying structure), or epidermis alone; the small wound in the skin is closed while the small misplaced particle grows, and finally becomes a small epithelial lined and filled cyst. Experimentally, Schweninger (and independently, the author, who enclosed bits of skin from the aglandular comb of a rooster), as well as Ribbert, Guszmann (lit.), Pels-Leusden, etc., produced traumatic epithelial cysts, by transplanting epithelium into spaces produced in the accessory structures; v. Kugelgen and Mori also, who modified the experimental means in various ways, produced such cysts (dermoidal atheromas), and epithelial cysts. Retterer believes all atheromas are due to a traumatic misplacement quite similar to that which occurs in Reverdin's epidermis cysts. The congenital dermoids of the skin originate from misplacement of rests of germinal ducts, or from the inclusion of skin or epithelial parts in the depths of the corium or the subcutaneous tissues. These dermoids are always sharply encapsulated, may reach the size of fists, and are found particularly frequent in the head, the nape of neck, the gluteal region, and other situations on the trunk, in which, during the embryonal time, misplacements of the ectoderm, can occur, or where there are fetal spaces and passage-ways covered with epidermis. The favoritism exhibited by dermoids is quite evident for the "lines of closure" of the body surface, and a frequent position is in old fissures. According to whether the enclosed piece of skin is poorly or better developed (Chiari), the wall shows microscopically the structure of the skin, perhaps merely

as an epithelium covering papillary bodies (epidermoid), or perhaps also the accessory structures (dermoide sensu strictiori). Internally in the dermoid, there are overgrowths of the wall in the form of pointed condylomas; benign epithelial overgrowths in dermoid cysts are not rare (Linser).

Following external irritations, traumata, etc., atheromas may ulcerate. Cutaneous horns arise from open dermoids. The development of carcinoma (squamous cell), has also been observed in atheromas, usually from those which were opened, and had been irritated over long periods of time (Krische, Mertens, Linser, Zesas and others). Differences of opinion exist as to whether carcinoma comes more frequently from epidermoids (Franke), or from follicular cysts (Kurtzahn, lit.). Frei (lit.), who

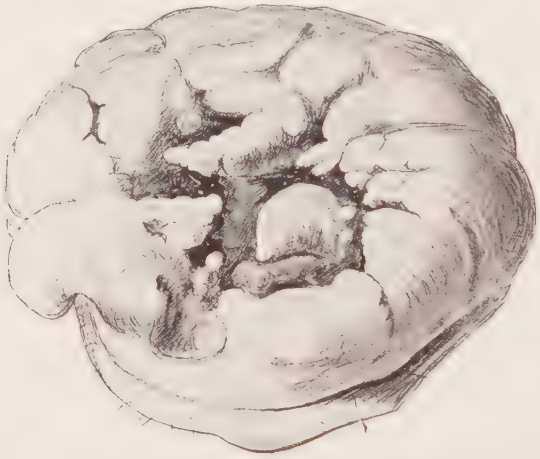


FIG. 1071.—Cornified carcinoma, ulcerated, from scalp, derived from an atheroma; woman 53 years old; nat. size.

described a case of the latter kind, believes that his case is the first certain one of its kind. (See under the breast p. 1802 for carcinomatous change in a closed dermoid, as described by the author.) Rarely there are extensive cornification and calcification in the epithelium and the connective tissue, whereby incrustated, almost bony, solid masses are formed.

4. For inflammations, see under acne and furuncle (p. 2104). For the sebaceous glands as the points of origin of carcinomas, see p. 2201; see also the experimental investigations concerning the regression of sebaceous glands, by Ribbert, Misumi, which explain to a certain extent, the origin of squamous epithelial carcinomas from the cells of sebaceous glands and of atheromas. For amyloid, see p. 2215.

Sweat Glands.— Pathologic anatomic changes in the sweat glands, the epithelium of which undergoes rapid post morten destruction, are little known, with the exception of the above mentioned miliaria (p. 2085),

suppuration in furunculosis, amyloid degeneration (p. 2215), sweat gland naevi (p. 2191), sweat gland adenomas (fig. 1059, p. 2200), and as the recently emphasized relationship to cysts in breasts and to mammary carcinoma (p. 1765). Nevertheless, there are various clinical conditions, in which, no doubt, finer changes of the secretory processes must occur; see Veil (lit.). Thus there are the very rare, complete absence (anidrosis); diminution of secretion (hypidrosis or oligidrosis), *e.g.*, in diabetes, myxedema, Bright's disease, etc.; increase of secretion (hyperidrosis), the latter unilateral in certain nervous diseases, or local (on the foot, in flat feet). We have already mentioned uridrosis, (p. 1358), the excretion of urinary substances in chronic kidney diseases and uremia, precipitation of urea on the skin after evaporation of the water in which it was dissolved. The excretion of medicaments (bromidrosis), and even blood in neuropathic individuals, also occurs. Recently Krompecher has called attention to the relationship of hypertrophy of the sweat glands with hyperplasia, and cystic dilatation of the glandular tubes to pregnancy. This is especially the case with the glands in the axillae, which secrete a characteristically smelling secretion, in all probability, a secondary sexual characteristic; Waelsch believes this increase in pregnancy is an increased function due to hormone stimulation; according to Rebaudi, it is a detoxification process of the organism; Krompecher recently found similar gland formations in benign tumors, and in carcinomas of the breast.

Of diseases of the hair, may be mentioned: (*a*) area celsi, or alopecia areata, porrigo s. tinea decalvans, characterized by total falling out of the hair, in circular or platter-shaped, sharply circumscribed situations. Usually there is a later replacement by new hair formed in the bottom of the old hair papillae just as the human hair is continuously changing. (Human hair grows about 0.4 millimeters daily. According to Stieda, in contrast to the older opinion, with every change of hair, there is new hair germinal tissue, from which a new hair grows from a new papilla. For more details see Fr. Merkel.) Graying and whitening of the hair (canities) is thus said to depend upon a change in the hair itself and is not merely a change in color. This is confirmed by Bloch, who also states, that in increasing age, when the hair becomes gray, the intensity of the Dopa reaction (p. 2189), becomes less, and when the hair is white to the bulbus, it is finally completely extinguished. Concerning changes in color, see also Choene (lit.). The influence of the nervous system on the pigment content is discussed by Nehl.

In the so-called malignant forms, the falling of the hair progresses quickly to total baldness (calvities). The etiology of alopecia areata, as well as of the small spotted baldness (areata parvimaculata), is still unknown. A trophoneurosis is thought of; Behrend (lit.) hypothecates a

causative local circulatory disturbance; possibly organized irritants, cocci, may play a role (Dreuw).

Concerning epidemic hair diseases, the causes of which are known, see under microsporia, p. 2151; trichophytes, p. 2150; favus, p. 2149; for more details and lit. see Sabouraud.

Other forms of alopecia are: alopecia adnata, congenital absence of hair (atrichie, hypotrichosis), very rare; at the same time the sebaceous glands may be less in number or completely undeveloped (Bettmann, Hoffmann (lit.), Waelsch); alopecia may also occur as a familial inheritable disease (Kraus, lit., Linzenmeier). In alopecia senilis (p. 2214) and praesenilis, the hair follicles and the sebaceous glands disappear; alopecia pityrodes (p. 2216), the symptomatic form, follows severe local diseases such as erysipelas, syphilitic efflorescences, roseolas, etc., but may also follow general diseases such as typhoid fever, syphilis (according to Sabouraud extraordinarily frequently), scarlet fever, measles, influenza—Pinkus, Zurhelle, Merian,—anemia, etc., and, interestingly enough, after severe accidents, and psychic traumas (Franck, Bretschneider); if the papilla is not destroyed, replacement of the hair may take place. Syphilitic alopecia occurs regularly in foci of small dimension (as large as pennies), irregular, very poorly outlined; a few hairs always remain in the spot; the skin in the focus is usually intact. As a rule, these situations become covered with hair after a longer or shorter period of time. (Concerning alopecia phenomenon in meningeal syphilis, see Gartner.)

For more details concerning the influence of diseases on the hair of the scalp, see Pinkus.

(*b*) **Trichorrhexis nodosa** (Kaposi), is a change in which the hairs show white points and nodules, produced by a longitudinal splitting, particularly in the beard and mustache. It is related to "point splitting" (trichoptilosis), and the result of drying out, in weakened nutritional circumstances (Kaposi, Michelson). Many believe that a parasite is the cause of the disease (some others not, Bruhns). Lit. by Sack.

(*c*) Hypertrophic development of the hair is known as hypertrichosis (hirsutism), which may be present at birth, or inheritable. Hair develops on situations otherwise covered by the lanugo hairs, and as a replacement of the disappeared fetal lanugo (Waldeyer); or, persistent lanugo hairs become larger (hypertrichosis lanuginea s. primaria, Mense, lit.), or, secondary hairs undergo an excessive enlargement. There may be differentiated: (1) hypertrichosis universalis, which is oberved in hypertrichotic hairy, or ape, or dog humans (P. Sarasin). The direction and arrangement correspond to those lines (fissure lines), and vertebrae, which have been described by Voigt and Langer for this situation of the hairs. (For teeth anomalies in hairy humans, see p. 584.) (2) Hypertrichosis partia-

lis, and heterochronic hypertrichosis (hair in situations normal for adults, but before adult time), *e.g.*, pubic and axillary hair in children, often bespeak premature puberty, (Herzog, and p. 1958); heterotopic hypertrichosis (hair in situations where otherwise there is lanugo hair), bearded women, often in those who have become sterile early, and also occasionally in those castrated (p. 1079), or in the so-called virago, the man-woman, *i.e.*, an individual with male hair attributes, and otherwise male habitus, who often show anomalies of the genitalia, thus an abnormally large clitoris (Torok), are all well known conditions. Bab believes hypertrichosis may be due to increased adrenal function after puberty, with changes in the heterosexual type. Even in hypertrichosis universalis such situations remain hairless, which, in normal humans, are entirely without hair (palms of the hand, soles of feet, prepuce, etc.). Lit. concerning this subject by Friedenthal. Naevi (naevi pilosi), are frequently very hairy, but this condition must be distinguished from actual hypertrichosis. Even in spina bifida occulta, there may be a circumscribed hypertrichosis in the sarcrolumbar region (p. 1976); in the phylogenetic sense, this has been looked upon as the rudiment of a tail, but according to W. Landau (lit.) and Schein, it is rather an inhibition of development, a persistence of fetal hair; according to others, it is the result of an increased growth stimulation in these parts exerted on the cells by abnormal influences (Broman). Acquired hypertrichosis has been observed following chronic irritations of the skin, in rare cases also in diseases of the spinal cord. For hypertrichosis in pineal tumors, see p. 1959. According to Halban, hair appears in the region of the linea alba during pregnancy (Jellinghaus). Changes in the hair are also observed in acromegaly and other disturbances of the endocrine glands (adrenals, etc.).

Diminished formation of hair, *i.e.*, persistence of fetal lanugo, is called hypertrichia or hypertrichosis lanuginosa s. primaria (Mense); these are seen in the second group of hypotrichotic, so-called hairy people. See p. 584 for tooth anomalies accompanying this condition, and also consult Parreidt.

In febrile and general diseases, the thickness of the hair is diminished (Matsuura). See Rottger concerning post mortem changes of the hair from long continued decomposition and destruction (auburn and blond color of previously dark brown or white hair).

Diseases of the Nails.—Hypertrophy of the nails is an increase in mass and extent above that of the normal. The nails may become like claws or like the horns of a steer (onychogryphosis).

Onychogryphosis occurs partly as a trophoneurotic disturbance in the course of nervous diseases, and partly from stasis and diminished nutrition of the skin, particularly in the varicose symptom-complex, etc. (p. 167).

In marasmus, the nails become dry, brittle, and sometimes hypertrophic.

An unguis incarnatus, ingrown nail, is caused by the growth of the nail into the cutis of the matrix, because the former is too broad; a paronychia is the inflammation of the bed of the nail. In onychia syphilitica, the bed of the nail is occupied by a papule or a gumma. A paronychia syphilitica may be an initial manifestation, or a papule or a pustule during the course of constitutional syphilis, or a gumma, and is localized to the matrix or the lateral sides of the nail. Gummas in this neighborhood ulcerate very frequently. According to whether the matrix and the bed of the nail have been destroyed in toto or only partly, the nail is totally or partially lost. The later substitution is rarely complete.

FIG. 1072.—Onychogryphos curved like a goat horn from large toe of man 59 years old; nat. size.

Onychomycosis favosa and trichophytina are discussed on pp. 2149 and 2150.

Onychomycosis saccharomycetica in the form of swelling, brittleness, yellow discoloration of the substance of the entire finger nail is described by Bourgeois in two women, the one an older woman suffering from diabetes.

In psoriasis vulgaris (p. 2097), there may be differentiated: (*a*) the so-called primary psoriasis of the nails (Schutz), with bow-like excavations on their surfaces; and (*b*) secondary psoriasis following the disease in the nail bed. There occur thickenings, cloudiness, brittleness, and lifting off of the nail itself at its distal part.

The records concerning its frequency vary between 9% (Heller, lit.) and 21% (Jadassohn-Radt).

In the very frequent leukonychia partialis (punctata, striata) and totalis (rare), the white discoloration is caused by air imbibition.

For the nails of albinos, see p. 2164. For scarlet fever lines on the finger nails, see p. 2076. Hour-glass shaped nails on club fingers, on p. 1115.

Concerning subungual exostoses, see p. 1199. Other tumors, chrondromas, and particularly sarcomas, are described by Wurmbrand.

APPENDIX

REFERENCES TO THE LITERATURE

Explanation of the Abbreviations

A. Chir. Archiv für klinische Chirurgie (begründet von *Langenbeck*).
A. d. H. Archiv der Heilkunde.
A. d. Méd. Archives de Méd. expérimentale et d'anatomie pathologique (begründet von *Charcot;* 1920 eingegangen).
A. d. Phys. Archives de Physiologie normale et pathologique.
A. f. A. Archiv für Anatomie und Physiologie.
A. f. D. Archiv für Dermatologie und Syphilis.
A. f. E. Archiv für Entwicklungsmechanik der Organismen (*W. Roux*).
A. f. e. P. Archiv für experimentelle Pathologie und Pharmakologie.
A. f. G. Archiv für Geburtshilfe und Gynäkologie.
A. f. Hyg. Archiv für Hygiene.
A. f. K. oder **Kh.** Archiv für Kinderheilkunde.
A. f. L. Archiv für Laryngologie und Rhinologie.
A. f. m. A. Archiv für mikroskopische Anatomie und Entwicklungsgeschichte.
A. f. O. Archiv für Ohrenheilkunde.
A. f. orth. u. U. Chir. Archiv für orthopädische und Unfallchirurgie.
A. f. Phys. Archiv für die gesamte Physiologie (begründet von *Pflüger*).
A. f. Psych. Archiv für Psychiatrie und Nervenkrankheiten.
A. f. Sch. u. Tr. Hyg. Archiv für Schiffs- und Tropenhygiene (*Mensee*).
A. f. V. Archiv für Verdauungskrankheiten (*Boas*).
A. gén. de Méd. Archives générales de Médecine.
A. I. M. Archives of Internal Medicine, Chicago.
A. J. American Journal of the medical sciences.
A. n. I. Wien. Arbeiten aus dem neurologischen Institut, Wien.
A. p. I. Homén. Arbeiten aus dem pathologischen Institut der Universität Helsingfors (Finnland), herausg. v. *Homén*, Berlin, Karger; Neue Folge bei Fischer, Jena.
A. p. I. von Baumgarten. Arbeiten aus dem pathol.-anat. Institut zu Tübingen, herausgegeben von *v. Baumgarten*.
A. p. sc. m. Archivio per le Scienze Mediche, fondato da *Bizzozero*.
Act. med. Scand. Acta medica scandinavica, früher N. M. A.
Ae. S. Z. Ärztliche Sachverständigen-Zeitung.
Amer. J. American Journal of the medical sciences.
An. A. Anatomischer Anzeiger.
An. H. Anatomische Hefte (*Merkel, Bonnet*).
Ann. Pasteur. Annales de l'Institut *Pasteur*, Paris.
B. Berliner klinische Wochenschrift; seit 1922 Klin. W.
B. d. sc. m. Bulletino delle scienze mediche, Bologna.
B. P. H. Ph. Bulletin of the Ayer clinical Laboratory of the Pennsylvania Hospital, Philadelphia.
B. z. A. etc. des Ohres etc. Beiträge zur Anatomie, Physiologie, Pathologie, Therapie des Ohres, der Nase und des Halses.
B. z. G. u. G. oder **B. z. G.** Beiträge zur Geburtshilfe und Gynäkologie (*A. Hegar*).

B. z. K. d. Inf. Beiträge zur Klinik der Infektionskrankheiten und zur Immunitätsforschung (*Brauer*).
B. z. K. d. T. Beiträge zur Klinik der Tuberkulose (*Brauer*).
B. z. kl. Ch. oder **Chir.** Beiträge zur klinischen Chirurgie (*v. Bruns*).
Bost. c. H. Medical and surgical reports of the Boston city Hospital.
Br. m. J. The British medical Journal.
C. Centralblatt für allgemeine Pathologie und pathologische Anatomie.
C. f. Bakt. Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten.
C. f. Ch. oder **Chir.** Centralblatt für Chirurgie.
C. f. G. Centralblatt für Gynäkologie.
C. f. H. u. G. Centralblatt für Herz- und Gefäskrankheiten (*Mönckeberg*).
C. f. i. M. Centralblatt für innere Medizin.
C. f. Schw. Correspondenzblatt für Schweizer Ärzte; 1919 in Wochenschrift, Schw. m. W., umgewandelt.
C. f. St. Centralblatt für Stoffwechselkrankheiten.
C. Grenz. Centralblatt für die Grenzgebiete der Medizin und Chirurgie.
C. M. Centralblatt für die medizinischen Wissenschaften.
C. U. Publications of Cornell University Medical College, New York City.
Columbia Un. Studies from the Department of Pathology, college of Phys. and Surgeons, Columbia Univ.
D. Deutsche medizinische Wochenschrift.
D. A. Deutsches Archiv für klinische Medizin.
D. M. Z. Deutsche Monatsschrift für Zahnheilkunde.
D. W. oder **Derm. W.** Dermatologische Wochenschrift.
D. Z. Chir. Deutsche Zeitschrift für Chirurgie.
D. Z. f. N. Deutsche Zeitschrift für Nervenheilkunde.
E. Ergebnisse der allgemeinen Pathologie (*Lubarsch, Ostertag*).
E. Anat. Ergebnisse der Anatomie und Entwickelungsgeschichte (*Merkel, Bonnet*).
E. Chir. Ergebnisse der Chirurgie und Orthopädie.
E. Hyg. Ergebnisse der Hygiene, Bakteriologie, Immunitätsforschung und experimentellen Therapie.
E. i. M. Ergebnisse der inneren Medizin und Kinderheilkunde.
F. Fortschritte der Medizin (begr. von *C. Friedländer*).
F. G. R. Fortschritte auf dem Gebiete der Röntgenstrahlen.
F. Z. Frankfurter Zeitschrift für Pathologie (begründet von *Eugen Albrecht*).
G. d. H. Gazette des hôpitaux, Paris.
Gann, Ergebnisse der Krebsforschung in Japan, Verlag Handaya, Tokio.
Guy's h. r. *Guy's* Hospital Report.
Hospitaltidende, Kopenhagen.
I.-D. oder **I.-Diss.** Inauguraldissertation.
J. oder **J. f. K.** oder **Kh.** Jahrbuch für Kinderheilkunde.
J. A. M. A. The Journal of the American Medical Association.
J. exp. M. The Journal of experimental medicine.
J. H. H. B. The Johns Hopkins Hospital Bulletin, Baltimore.
J. H. H. R. The Johns Hopkins Hospital Reports, Baltimore.
J. M. R. The Journal of Medical Research, Boston.
J. of Anat. Journal of Anatomy and Physiology.
J. P. B. The Journal of Pathology and Bacteriology.
Klin. W. Klinische Wochenschrift, seit 1922 Fortsetzung der Berliner klin. W. u. Therapeutischen Halbmonatshefte.
L. P. m. La Presse médicale.
Lav. Ist. Barbacci. Lavori dell' Istituto di anat. patol. dir. dal Prof. *Barbacci*, Siena.
Lav. Ist. Cesaris-Demel. Lavori dell' Istituto di anat. patol. dir. dal Prof. *Cesaris-Demel*, Pisa.

Lav. Ist. Foà. Lavori dell, Istituto di anat. patol. dir. dal Prof. *Pio Foà*, Torino.
Lav. Ist. Martinotti. Lavori dell' Istituto di anat. patol. dir. dal Prof. *Martinotti*, Bologna.
M. Münchener medizinsche Wochenschrift.
M. f. G. Monatsschrift für Geburtshilfe und Gynäkologie (*Martin* und *Sänger*).
M. f. K. Monatsschrift für Kinderheilkunde (*Ashby, Czerny* u. a.).
M. f. O. Monatsschrift für Ohrenheilkunde u. Laryngo-Rhinologie.
M. f. U. Monatsschrift für Unfallheilkunde.
M. G. Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie.
M. K. oder **M. Kl.** Medizinische Klinik, Wochenschrift für prakt. Ärzte.
M. K. G. Mitteilungen aus dem Kaiserlichen Gesundheitsamt, Berlin.
M. p. I. København. Meddelelsen fra Københavns Univ. patol. anat. Institut (*Fibiger*).
M. St. Hamburg. Mitteilungen (oder Jahrbuch) aus den (oder der) Hamburgischen Staatskrankenanstalten.
Ma. G. H. Publications of the Massachusetts General Hospital, Boston.
N. M. Norsk Magazin for Laegevidenskaben, Kristiania.
N. M. A. Nordiskt medicinskt Arkiv, Stockholm.
N. Z. Neurologisches Zentralblatt.
P. Prager medizinische Wochenschrift.
P. Cornell Univ. Publications of Cornell Univ. Med. College, New York City.
P. New York. Proceedings of the New York Pathological Society.
Path. oder **Pathologica.** Pathologica, rivista quindicinale.
Path. G. Verhandlungen der deutschen Pathologischen Gesellschaft.
Path. G. Japan. Verhandlungen der japanischen pathologischen Gesellschaft.
Pennsylvania. Un. Pennsylvania Med. Bull.
R. de Chir. Revue de Chirurgie, Paris.
R. méd. Revue médicale de la Suisse romande.
Rock. Inst. oder **Stud. fr. R. Inst. f. med. R.** Studies from the Rockefeller Institute for medical research, New York.
S. kl. V. Sammlung klinischer Vorträge (begründet von *Volkmann*).
Schw. m. W. Schweizerische medizinische Wochenschrift (früher C. f. Schw.).
Sperimentale. Lo Sperimentale (Archivio di Biologia normale e patologica).
Stud. Path. Entw. Studien zur Pathologie der Entwickelung, von *R. Meyer* und *E. Schwalbe*.
Surgery, G. and O. Surgery, Gynecology and Obstetrics, Official Journal of American College of Surgeons.
T. G. Nederl. Tijdschrift voor Geneeskunde.
Tr. Inst. Stilling. Travaux de l'Inst. pathol. de Lausanne (*H. Stilling*).
V. Vierteljahrsschrift für gerichtliche Medizin und öffentliche Sanitätspflege.
V. A. *Virchows* Archiv für pathologische Anatomie und Physiologie und für klinische Medizin.
V. H. *Virchow-Hirsch's* Jahresbericht.
W. oder **W. m. W.** Wiener medizinische Wochenschrift.
W. A. Wiener Archiv für innere Medizin.
W. klin. R. Wiener klinische Rundschau.
W. klin. W. oder **kl. W.** Wiener klinische Wochenschrift.
W. m. P. Wiener medizinische Presse.
Z. B. Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie (begründet von *Ziegler*).
Z. e. P. Zeitschrift für experimentelle Pathologie und Therapie.
Z. f. a. A. Zeitschrift für angewandte Anatomie und Konstitutionslehre (*Tandler*).
Z. f. A. u. E. Zeitschrift für Anatomie u. Entwicklungsgeschichte, Abt. I von Z. f. g. A.
Z. f. e. M. Zeitschrift für die gesamte experimentelle Medizin.
Z. f. G. Zeitschrift für Geburtshilfe und Gynäkologie.
Z. f. g. A. Zeitschrift für die gesamte Anatomie.
Z. f. g. N. u. Psych. Zeitschrift für die gesamte Neurologie und Psychiatrie (begründet von *Alzheimer* und *Lewandowsky*).

Z. f. H. Zeitschrift für Heilkunde.
Z. f. Hyg. Zeitschrift für Hygiene und Infektionskrankheiten.
Z. f. I. H. Zeitschrift für Infektionskrankheiten, parasit. Krankheiten und Hygiene der Haustiere.
Z. f. Imm. Zeitschrift für Immunitätsforschung u. experim. Therapie.
Z. f. K. Zeitschrift für Krebsforschung.
Z. f. Kh. Zeitschrift für Kinderheilkunde.
Z. f. kl. M. Zeitschrift für klinische Medizin.
Z. f. L. Zeitschrift für Laryngologie und Rhinologie und ihre Grenzgebiete.
Z. f. N. oder **D. Z. f. N.** Deutsche Zeitschrift für Nervenheilkunde.
Z. f. Ohr. Zeitschrift für Ohrenheilkunde und für die Krankheiten der Luftwege.
Z. f. R. Zeitschrift für Röntgenkunde.
Z. f. T. Zeitschrift für Tuberkulose.
Z. f. Urol. Zeitschrift für Urologie.
Z. f. urol. Chir. Zeitschrift fur urologische Chirurgie.

The expression (lit.) means that the reference cited contains the collected literature of the subject.

When the expression, "lit. in appendix" is used in the text, it means, see the lit. cited in the appendix.

The heavy type refers to the pages of the book.

Weekly journals are cited, first by year, then the week number, for instance, D. 1910, 1, is Deutsche Med. Wochenschrift 1910, #1. Other journals, archives, etc., are cited first by volume, then by year, *e.g.*, Z. B. 65, 1919 is Ziegler's Beitrage, volume 65, 1919.

Heart

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609 *Löffler*, l. c. — *M. Neisser*, Hyg. Rundschau 1903, 14. — *Kolle* u. *Schlossberger*, Z. f. Hyg. 90, 1920. — *Brieger* u. *C. Fränkel*, B. 1890. *Petruschky*, A. a. d. Path. Inst. Tübingen VI, 1908, Festschr. f. *v. Baumgarten*. — *Schweriner*, Z. f. Hyg. 88, 1919. — *Büsing*, Z. f. Hyg. 57, 1907. — *Flügge*, l. c. p. 355. — *Germano*, Z. f. Hyg. 25. Bd. — *Daut*, J. f. K. 48, 1898. — *Joannovis*, W. kl. W. 1919, 9, *Bingels* Ansicht ist zu verwerfen. **610** *Hoffmann* s. bei *Neisser*, l. c. p. 476. — *Löffler*, l. c. p. 476. — *M. Neisser*, l. c. p. 476. — *Roux*, *C. Fränkel* s. bei *Uhthoff*, B. 1899.

— *Klinger* u. *Schoch*, Z. f. Hyg. 80, 1915. — *M. Neisser*, l. c. — *Scheller*, in *Kolle-Wassermanns* Handb. II, Ergsbd. — *Gins*, D. 1913, 11, S. 502. — *Mühsam*, B. 1916, 24, Diphth.ähnl. B. im Zahnbelag. — *Landau*, B. 1916, 26, Diphth.ähnl. B. im Zahnbelag, Rachen, Tonsillenabstrich. — *Axenfeld*, Univ.-Programm, Freiburg 1905 u. *Kolle-Wassermanns* Handb. Bd. 3. **611** *Heubner*, S. kl. V., Nr. 322, 1888, J. f. K. 1889; Kongr. f. inn. Med. VIII, 1889; J. f. K. 1890. *Weigert*, V. A. 70, 1877 u. 79, 1880. — *Heubner*, l. c. p. 477. **612** *Graetz*, C. f. Bakt. Orig. 84, 1920, Heft 6. **613** *Róna*, Zur Ätiol. u. Pathog. der *Plaut-Vincent*schen Angina, der Stomakace, der Stomatitis gangr. idiopath., bzw. der Noma, der Stomatitis mercurial. gangraen. un der Lungengangrän, A. f. D. 74, 1905. — *Babes*, l. c. p. 428. — *Blühdorn*, D. 1911, 25. — *Scheier*, B. 1909, 50, S. 2268, Lymph nodes slightly affected with Pl.-V. angina, but usually markedly so in primary affections. — *Gerber*, l. c. p. 428. — *Reiche*, Beitr. z. Kenntnis der Angina exsudativa ulcerosa, Mitt. Hamb. Staatskr. V, 1905. — *Baron*, Deutsch. Ges. f. Kinderhlkde., Dresden 1907. — *Rothwell*, J. A. M. A., 4. Juni 1910, 2 Fälle dieser Art, endeten mit Genesung. — *Graetz*, l. c. p. 479. — *Huebschmann*, M. 1917, 3. — *Fahr*, V. A. 221, 1916. — *Hochhaus*, V. A. 124, 1891. — *Sörrensen*, Z. f. Hyg. 81, 1916. — *Hotzen*, Z. B. 60, 1915. — *Huebschmann*, l. c. p. 46. **614** *Weinert*, Wund- u. Narbendiphtherie, M. 1919, 9. — *Nieter*, M. 1919, 9, in 497 cases, 58.6% gave positive true D. B. findings. — *Hock*, M. 1919, 23. — *Frankenthal*, A. Chir. 117, 1921, Mixed infection, espec. strept. erysip. plays a part. — *Wieting*, C. f. Chir. 1921, 32, considers the name "wound diphtheria" incorrect. Diph. B. are not important, specific therapy fails; s. auch *Hilgenreiner*, D. Z. Chir. 170, 1922, Lit. — *Rohde*, M. 1921, 8. — *J. Koch*, Scharlacherreger, E. XIII, 1, 1909. — *Jochmann*, Bakterienbefunde bei Scharlach u. ihre Bedeutung f. d. Krankheitsprozess, Z. f. kl. M. 56, 1905. — *Buloch-Schmorl*, Z. B. 16, 1894. **615** *Burckhardt-Merian*, S. kl. V. 182. *Aschoff*, Lehrb., 4. Aufl. 1919, S. 779. — *Kretz*, M. G. 17, 1907, S. 8. **617** *Most*, A. Chir. 61, 1900. — *Lexer*, A. Chir. 54. — *Kien*, Z. f. Ohrenhlkde. 39, 1901. — *Most*, l. c. — *Beitzke*, Path. G. 12, 1908. — *Bartels*, l. c. p. 130. **618** *Orth*, Arb. a. d. Path. Inst. Göttingen 1893. — *Deichert*, V. A. 141, 1895. — *Nösske*, D. Z. Chir. 66, 1903. — *Schweitzer*, Knorpel- u. Knochenbildg. in den Gaumenmandeln, I.-D. Freiburg 1905, Lit. — *Lubarsch*, V. A. 177, 1904.

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Vagina, Vulva

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Nervous System

A. Diseases of the Meninges

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Heavy face type denotes an illustration.

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Heavy face type denotes an illustration.

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Heavy face type denotes an illustration.

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